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THE TRANSACTIONS

OF THE

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OF EDINBURGH

VOL. XXVII.—NEW SERIES

SESSION 1907-1908

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PREFACE

THE present Volume is the *Twenty-seventh* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session Extra Meetings were held for the exhibition of Patients, Pathological Specimens, Instruments, etc. It is hoped that such Meetings will materially increase the usefulness of the Society.

Two of the Clinical Meetings were held in the Royal Infirmary: both were very successful.

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

A general Index to the first Twenty Volumes of the New Series was published in 1902. It is uniform with the other Volumes.

WILLIAM CRAIG,
Editor.

September 1908.

Medico-Chirurgical Society of Edinburgh.

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445	Hector Thompson Wilson, M.D., Ch.B., *Harrogate*,	1904
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	James William Somerville, M.D., D.P.H., *Saskatchewan*,	1905
	William H. Myers, M.D., *Indiana, U.S.A.*,	1906
	Herbert Stephen, L.R.C.P.E., M.R.C.S. Eng., *Bristol*,	1906
450	Frederick Dimmer, M.D., D.P.H., *Tewkesbury*,	1906
	Alfred Nolan Peck, M.D., M.R.C.P.E., *Chatham*,	1907
	William M'Kelvy, L.R.C.P.E., L.R.C.S.E., *Tokomaru*,	1907

ORDINARY MEMBERS.

ARRANGED ALPHABETICALLY.

(*a*) Members who pay the Annual Subscription of Ten Shillings.

	Dr K. Abernethy, 18 St Colme Street,	1892
	Dr J. O. Affleck, 58 Heriot Row,	1871
	Dr A. C. Allardice, 49 Melville Street,	1898
	Dr R. S. Aveling, 74 Great King Street,	1887
5	Dr J. Ivy Avery, Dunedin,	1898
	Dr James Ballantyne, 2 Ainslie Crescent,	1869
	Dr J. F. Barnes, 6 Hamilton Terrace,	1894
	Dr J. L. Bathgate, 15 Manchester Road,	1904
	Dr Harold S. Bathurst, Lucknow,	1899
10	Dr J. W. Beattie, 31 Melville Street,	1885
	Dr A. H. Bennett, Egham, 4 Clarence Square,	1881
	Dr Lewis Bruce, 13 Temple Park Street,	1905
	Dr J. H. Montgomerie Bell, Fort Matanzas, Florida, Care of Rees,	1905
	Dr Joseph Bell, 2 Melville Crescent,	1882
15	Dr G. A. Berry, 31 Drumsheugh Gardens,	1885
	Dr W. L. Bevis, 2 Grosvenor Street,	1877
	Dr Robert H. Blackie, 11 Mayfield Gardens,	1885
	Dr G. P. Bonthron, 73 Drumsheugh Gardens,	1888
	William Boog, Esq., 2 Melville Street,	1888
20	Dr Felix M. Bowie, 36 Great King Street,	1901
	Dr F. D. Boyd, C.M.G., 22 Melville Street, *Sydney*,	1891
	Dr Patten Brotherwell, 23 Drumsheugh Gardens, *Edinburgh*,	1876
	Dr Lewis Brotherwell, 24 Walker Street,	1900
	Dr N. T. Brown, 9 Drumsheugh Gardens,	1886

		Date of Admission
25	Dr A. J. Brock, 27 Braid Crescent,	1907
	Dr Henry Grey Brown, 47 Evan Street, Stonehaven,	1905
	Dr J. J. Graham Brown, 3 Chester Street,	1878
	Dr J. Murdoch Brown, 9 Walker Street,	1885
	Dr R. Dods Brown, Royal Asylum, Edinburgh,	1908
30	Dr Alexander Bruce, 8 Ainslie Place,	1883
	Dr A. H. Buchan, 2 Dean Terrace,	1899
	Dr Buist, 1 Clifton Terrace,	1877
	Dr T. M. Burn-Murdoch, 14 Charlotte Square,	1886
	Dr J. Burnet, 6 Glengyle Terrace,	1907
35	Professor Francis M. Caird, 13 Charlotte Square,	1883
	Dr H. L. Calder, 60 Leith Walk, Leith,	1884
	Dr James Cameron, 13 Fettes Row,	1895
	Dr Malcolm Campbell, Brechin,	1904
	Dr A. N. S. Carmichael, 2 Merchiston Avenue,	1902
40	Dr Edward Carmichael, 21 Abercromby Place,	1887
	Dr E. W. Scott Carmichael, 32 Rutland Square,	1901
	Dr George Scott Carmichael, 25 Braid Road,	1903
	Dr J. Carmichael, 22 Northumberland Street,	1870
	Dr George J. R. Carruthers, 4A Melville Street,	1901
45	C. W. Cathcart, Esq., 44 Melville Street,	1883
	Dr J. G. Cattanach, 3 Alvanley Terrace,	1895
	Dr T. F. S. Caverhill, 6 Manor Place,	1883
	George L. Chiene, Esq., 23 Alva Street,	1901
	Professor John Chiene, C.B., 21 Alva Street,	1867
50	Dr B. R. Craig Christie, 32 Inverleith Gardens,	1907
	Dr Church, 36 George Square,	1876
	Dr A. W. Greenhorn Clark, 24 Braid Crescent,	1908
	Dr Clouston, 26 Heriot Row,	1861
	Dr John D. Comrie, 7 S.-E. Circus Place,	1902
55	J. M. Cotterill, Esq., 24 Manor Place,	1878
	Dr John Craig, 71 Bruntsfield Place,	1900
	Dr William Craig, 71 Bruntsfield Place,	1869
	Dr D. M. Crichton, Burgh House, Maryhill, Glasgow,	1906
	Dr David Halliday Croom, 17 Alva Street,	1906
60	Professor Sir John Halliday Croom, 25 Charlotte Square,	1870
	Dr G. Matheson Cullen, 50 Minto Street,	1892
	Dr John Cumming, 70 Bruntsfield Place,	1894
	Professor D. J. Cunningham, 18 Grosvenor Crescent,	1903
	Dr T. B. Darling, 13 Merchiston Place,	1887
65	Dr G. D. Darlington, 10 Ardmillan Terrace,	1898
	Dr G. A. Davies, 7 West Holmes Gardens, Musselburgh,	1906
	Dr M. Dewar, 24 Lauriston Place,	1885
	Dr Thomas W. Dewar, Kincairn, Dunblane,	1906
	Dr J. T. Dickie, 37 Lauriston Place,	1900
70	Dr D. Elliot Dickson, Hill Crescent, Lochgelly, Fife,	1906
	Dr George Dickson, 9 India Street,	1884
	Dr George Dickson, 14 Ardmillan Terrace,	1901
	Dr W. E. Carnegie Dickson, 92a Findhorn Place,	1902
	Dr C. E. Douglas, Cupar-Fife,	1894
75	Dr Kenneth M. Douglas, 19 Chester Street,	1888
	Dr W. B. Dow, Dunfermline,	1879
	J. W. Dowden, Esq., 48 Manor Place, *Treasurer*,	1893
	Dr W. B. Drummond, 2 Braidburn Terrace,	1900
	Dr James K. Drysdale, 22 Coltbridge Terrace,	1901
80	Dr Henry J. Dunbar, 3 Hampton Terrace,	1906
	Dr John A. H. Duncan, 32 Morningside Drive,	1895
	Dr Kirk Duncanson, 22 Drumsheugh Gardens,	1871
	Dr H. M. Dunlop, 20 Abercromby Place,	1883
	Dr J. C. Dunlop, 33 Chester Street,	1892
85	Dr John Eason, 58 Northumberland Street,	1900
	Dr William Elder, 4 John's Place, Leith,	1892

		Date of Admission.
	Dr J. Haig Ferguson, 7 Coates Crescent,	1885
	Dr Thomas Finlay, 7 Mary's Place,	1903
	Dr W. A. Finlay, 50 Trinity Road,	1875
90	Dr Andrew F. Fleming, St John's Road, Corstorphine,	1905
	Dr R. A. Fleming, 10 Chester Street,	1890
	Dr A. B. Flett, 60 George Square,	1903
	Dr W. E. Foggie, 4 Springfield, Dundee,	1907
	Dr A. Dingwall Fordyce, 19 Coates Crescent,	1901
95	Dr W. Fordyce, 20 Charlotte Square,	1907
	Dr L. Fourie, Chalmers Hospital, Edinburgh,	1908
	Dr J. S. Fowler, 68 Northumberland Street,	1895
	Dr John Fraser, 13 Heriot Row,	1878
	Dr J. Hossack Fraser, Fernfield, Bridge of Allan,	1895
100	Dr J. S. Fraser, 36 Moray Place,	1905
	Prof. Sir Thomas R. Fraser, 13 Drumsheugh Gardens,	1865
	Dr W. Ernest Frost, 6 Athol Place,	1904
	Dr F. Gardiner, 9 George Square,	1900
	Dr Garland, 53 Charlotte Street, Leith,	1873
105	Dr A. Campbell Geddes, Keelhouse, Gateland Road,	1904
	Dr J. H. Gibbs, 12 Coates Crescent,	1905
	Dr G. A. Gibson, 3 Drumsheugh Gardens,	1880
	Dr A. B. Giles, 4 Palmerston Place,	1893
	Dr Alexander Goodall, 7 Mayfield Road,	1901
110	Dr D. J. Graham, 26 Rutland Street,	1897
	Dr F. M. Graham, 10 Mayfield Gardens,	1894
	Dr J. Taylor Grant, 3 Manor Place,	1898
	Dr J. Allan Gray, 107 Ferry Road,	1879
	Dr John L. Green, 23 Minto Street,	1904
115	Professor Greenfield, 7 Heriot Row,	1886
	Dr G. L. Gulland, 11 Chester Street,	1888
	Dr James A. Gunn, 18 Morningside Drive,	1906
	Dr A. C. Guthrie, 21 Pilrig Street,	1890
	Dr William Guy, 11 Wemyss Place,	1890
120	Dr William Haldane, Viewforth, Bridge of Allan,	1880
	Dr George Halley, 7 Airlie Place, Dundee,	1902
	Dr D. Berry Hart, 5 Randolph Cliff,	1886
	Dr James Harvey, 7 Blenheim Place,	1893
	J. W. B. Hodgson, Esq., 6 Chester Street,	1883
125	Dr George Hunter, 33 Palmerston Place, *Vice-President*,	1876
	Dr David Hutkie, Moffat,	1899
	Dr J. Hutcheson, 44 Moray Place	1890
	Dr R. W. Inkster, 16 Windsor Street,	1895
	Dr Alex. Innes, 14 Randolph Crescent,	1877
130	Dr Allan Jamieson, 35 Charlotte Square,	1870
	Dr Hugh Jamieson, 1 Strathearn Road,	1889
	Dr J. Boyd Jamieson, 43 George Square,	1900
	Dr G. M. Johnston, 7 Wellington Place, Leith,	1888
	Dr R. J. Johnston, 18 George Square,	1877
135	Dr R. M'Kenzie Johnston, 2 Drumsheugh Gardens,	1883
	Dr R. W. Johnstone, 13 Torphichen Street,	1907
	Dr C. Kennedy, 5 Salisbury Road,	1886
	Dr C. B. Ker, City Hospital, Colinton Road,	1894
	Dr George Kerr, 6 St Colme Street,	1885
140	Dr J. Lamond Lackie, 1 Randolph Crescent,	1907
	Dr J. H. A. Laing, 11 Melville Street,	1889
	Dr H. G. Langwill, 4 Hermitage Place, Leith,	1899
	Dr David Lawson, Nordrach-on-Dee, Banchory,	1901
	Dr H. A. Lediard, Corstorphine,	1899
145	Dr Edwin M. Lithgow, 8 Alva Street,	1901
	Dr John D. Lithgow, 8 Alva Street,	1901
	Professor Harvey Littlejohn, 11 Rutland Street,	1889
	Emeritus Professor Sir Henry D. Littlejohn, 24 Royal Circus,	1853

		Date of Admission
	Dr R. Cranston Low, 6 Castle Terrace,	1906
150	Dr T. Davey Luke, 6 Belford Terrace,	1900
	Dr R. A. Lundie, 55A Grange Road,	1883
	Dr W. Leslie Lyall, 8 Murrayfield Gardens,	1902
	Dr D. C. A. M'Allum, 11 Greenhill Gardens,	1900
	Dr P. M'Bride, 16 Chester Street,	1879
155	Dr Angus Macdonald, 27 Manor Place,	1902
	Dr Stuart M'Donald, 197 Newhaven Road,	1901
	Dr John M'Gibbon, 22 Heriot Row,	1899
	Dr C. W. MacGillivray, 15 Charlotte Square, *Vice-President*,	1877
	Dr A. M. M'Intosh, 30 East Claremont Street,	1904
160	Dr G. Mackay, 74 Bruntsfield Place,	1878
	Dr George Mackay, 20 Drumsheugh Gardens,	1887
	Dr W. Leslie Mackenzie, 1 Stirling Road, Trinity,	1895
	Dr Malcolm M'Larty, 7 Bellevue Place,	1900
	Dr John Macmillan, 48 George Square,	1897
165	Dr J. C. G. Macnab, The Towers, Dysart,	1904
	Dr Alexander H. Macpherson, Chiftonville, Blackhall,	1901
	Dr John Macpherson, 8 Darnaway Street,	1891
	Dr G. Douglas M'Rae, Glengall House, Ayr,	1904
	Dr John MacRae, Lynwood, Murrayfield,	1893
170	Dr Alexander M. Malcolmson, St John's Road, Corstorphine,	1905
	Major D. G. Marshall, I.M.S., 25 Buckingham Terrace,	1905
	Dr A. A. Matheson, 41 George Square,	1901
	Dr Roderick M. Matheson, 33 Buccleuch Place,	1902
	Dr Edwin Matthew, 6 Hampton Terrace,	1900
175	Dr J. B. Mears, 26 Nile Grove,	1907
	Dr J. Halley Meikle, 12 Midmar Gardens,	1906
	Dr D. Menzies, 20 Rutland Square,	1878
	A. Miles, Esq., 20 Walker Street,	1892
	Dr J. Mill, 188 Ferry Road,	1886
180	A. G. Miller, Esq., 8 Garscube Terrace,	1867
	Dr W. H. Miller, 51 Northumberland Street,	1895
	Dr W. Morrison Milne, 10 Newington Road,	1905
	Sir Arthur Mitchell, 34 Drummond Place,	1859
	Dr John Mowat, 5 Hope Park Terrace,	1885
185	Dr T. Goodall Nasmyth, 27 Palmerston Place,	1884
	Dr H. Oliphant Nicholson, 20 Manor Place,	1900
	Dr John Orr, 6 Strathearn Road,	1895
	Dr W. Basil Orr, 13 Braid Road,	1893
	Dr G. Keppie Paterson, 19 Albany Street,	1889
190	Dr J. V. Paterson, 5 Castle Terrace,	1900
	Dr Simla Paterson, 147 Bruntsfield Place,	1904
	Dr P. Pattison, Parkhill, Colinton,	1907
	Dr C. Balfour Paul, 17 Walker Street,	1904
	Dr C. M. Pearson, 14 Manor Place,	1902
195	Dr H. A. Peddie, 24 Palmerston Place,	1893
	Dr R. W. Philip, 45 Charlotte Square,	1883
	Dr J. H. Harvie Pirie, 13 Alva Street,	1905
	Dr John Pirie,	1902
	Dr Playfair, 5 Melville Crescent,	1874
200	Dr Frederick Porter, 65 Morningside Road,	1904
	Dr W. G. Porter, 16 Manor Place,	1907
	Dr T. Proudfoot, 30 Lauriston Place,	1889
	Dr Harry Rainy, 16 Great Stuart Street,	1896
	Dr Douglas G. Reid, Finkenberg, St Barnabas Road, Cambridge,	1905
205	Dr James Ritchie, 22 Charlotte Square, *President*,	1873
	Dr James Ritchie, 10 Succoth Gardens,	1907
	Dr L. C. Peel Ritchie, 7 Alva Street,	1904
	Dr W. T. Ritchie, 9 Atholl Place,	1900
	Dr George M. Robertson, Tipperlinn House, Morningside Place,	1890
210	Dr Robert Robertson, 26 Royal Circus,	1901

		Date of Admission.
	Dr William Robertson, 11 Denham Green Terrace, Trinity,	1902
	Dr W. Ford Robertson, 48 Northumberland Street,	1895
	Dr W. G. Aitchison Robertson, 2 Maxwell Gardens,	1892
	Dr R. M. Ronaldson, 17 Morningside Road,	1895
215	Dr T. K. Ronaldson, 8 Charlotte Square,	1877
	Dr A. Aitken Ross, 8 Newington Road,	1902
	Dr William Russell, 3 Walker Street,	1884
	Dr J. M. Rutherford, Morningside Royal Asylum,	1900
	Dr E. G. Salt, 59 George Square,	1895
220	Dr James Scott, 43 Minto Street,	1895
	Dr Theodore Shennan, 95 Mayfield Road	1897
	Dr W. C. Sillar, 46 George Square,	1900
	Emeritus Professor Sir Alexander R. Simpson, 52 Queen Street,	1850
	Dr F. D. Simpson, 7 Kew Terrace,	1905
225	Dr G. T. Balfour Simpson, 50 Melville Street,	1900
	Dr John W. Simpson, 19 Lansdowne Crescent,	1904
	Dr Arthur H. H. Sinclair, 5 Walker Street,	1900
	A. A. Scot Skirving, Esq., C.M.G., 8 Randolph Crescent,	1897
	Dr A. T. Sloan, 22 Abercromby Place,	1885
230	Dr Andrew Smart, 37 Queen's Avenue, Blackhall,	1865
	Dr James Smith, 4 Prunton Place,	1891
	Dr John Smith, 11 Wemyss Place,	1856
	Dr J. Cormack Smith, 9 Brunton Place,	1890
	Dr Arthur J. de Sequeira Snell, Dunloe, Tranent,	1905
235	Dr J. W. Stevens, 13 Manor Place,	1903
	Dr John Stevens, 78 Polwarth Terrace,	1892
	Dr Norman L. Stevenson, 7 Alva Street,	1902
	Dr Ian Struthers Stewart, 27 Castle Terrace,	1900
	Dr J. S. Stewart, 15 Merchiston Place,	1885
240	Dr Robert Stewart, 25 George Square,	1892
	Dr William Stewart, 140 Ferry Road, Leith,	1880
	H. J. Stiles, Esq., 9 Great Stuart Street,	1882
	Dr John Strachan, Dollar,	1897
	John W. Struthers, Esq., 5 Lynedoch Place,	1901
245	W. J. Stuart, Esq., 9 Chester Street,	1902
	Dr William G. Sym, 12 Alva Street,	1880
	Dr W. Taylor, 12 Melville Street,	1874
	Dr W. Macrae Taylor, 12 Melville Street,	1900
	Dr C. H. Thatcher, 8 Melville Crescent,	1878
250	Dr R. Thin, 25 Abercromby Place,	1876
	Alexis Thomson, Esq., 30 Drumsheugh Gardens,	1887
	Dr H. Torrance Thomson, 12 Fleshhouse Place,	1903
	Dr John Thomson, 14 Cortes Crescent,	1887
	Dr George Thyne, 13 Brunswick Street	1904
255	Dr T. J. Thyne, 16 Kinnaird Crescent,	1898
	Dr John Tod, 69 Ferry Road, Leith,	1895
	Sir John Batty Tuke, M.P., 20 Charlotte Square,	1884
	Dr T. J. Turnbull, 6 Randolph Place,	1896
	Dr Dawson Turner, 37 George Square,	1896
260	Dr Logan Turner, 27 Walker Street,	1902
	Henry Wade, Esq., 32 Alva Street,	1901
	Dr Alexander Walker, 12 Lauder Road,	1897
	Dr Norman Walker, 7 Manor Place,	1891
	D. Wallace, Esq., C.M.G., 29 Charlotte Square, *Secretary*,	1882
265	Dr David Waterston, 1 Castle Place,	1903
	Dr Douglas Chalmers Watson, 22 Castle Crescent,	1884
	Dr Charles Herbert Watson,	1905
	Dr W. de Watteville, Kingussie,	1902
	Dr A. D. Webster, 18 Minto Street,	1884
270	Dr J. H. Douglas Webster, 20 Newington Road,	1907
	Dr James Watson, 53 Inverleith Row,	1901
	J. T. Wilkie, Esq., 4 Hamilton Place,	1883

		Date of Admission.
	Dr A. Murray Wood, 180 Ferry Road, Leith,	1907
	Dr Russell E. Wood, 9 Darnaway Street,	1883
275	Professor John Wyllie, 44 Charlotte Square,	1868
	Dr P. A. Young, 25 Manor Place,	1870

(*b*) Members Exempted under Rule V. from paying the Annual Subscription.

	Dr D. Aitken, 152 *Packington Street, Islington, London, N.*,	1887
	Dr H. de M. Alexander, *Aberdeen District Asylum, Aberdeen*,	1901
	Dr D. H. Anderson, 143 *Abbey Road, Barrow-in-Furness*,	1887
280	Dr J. A. Armitage, 58 *Waterloo Road. Wolverhampton*,	1887
	Dr W. Badger, *Penicuik*,	1882
	Dr Edwin Baily, *Oban*,	1883
	Dr Andrew Balfour, *Khartoum*,	1895
	Dr J. H. Balfour, *Portobello*,	1881
285	Dr Alexander Ballantyne, *Dalkeith*,	1872
	Dr Noel D. Bardswell, *Midhurst Sanitorium, Sussex*,	1901
	Dr W. H. Barrett, 29 *Park Crescent, Hesketh Park, Southport*,	1890
	Sir George T. Beatson, K.C.B., 7 *Woodside Crescent, Glasgow*,	1895
	Professor James M. Beattie, M.B., *The University, Sheffield*,	1904
290	Surgeon-Captain C. H. Bedford, *Bengal Army, care of W. Watson & Co.*, 28 *Apollo Street, Bombay*,	1889
	Dr Charles Begg, 62 *Pulteney Street, Bath*,	1900
	Dr Benjamin D. C. Bell, *Kirkwall*,	1889
	Dr G. J. H. Bell, *Bengal Army*,	1884
	Dr G. H. Bentley, *Kirkliston*,	1877
295	Professor Richard J. A. Berry, M.D., *University of Melbourne*,	1893
	Dr J. S. Beveridge, *Melton Constable, Norfolk*,	1861
	Dr J. W. Black, 15 *Clarges Street, Piccadilly, London, W.*,	1865
	Dr P. G. Borrowman, *Galvalmore, Crieff*,	1893
	Dr D. G. Braidwood, *Halkirk, Caithness*,	1889
300	Dr David Brodie, *Singlsby Villa, Royal Park Road, Finchley, N.*,	1865
	Dr Andrew Brown, 1 *Bartholomew Road, Kentish Town, London, N.W.*,	1884
	Dr John Brown,	1878
	Dr J. Macdonald Brown, 2 *Fragnall, Hampstead, London, N.W.*,	1883
	Dr William Brown, *Braemar*,	1904
305	Dr Lewis C. Bruce, *The Asylum, Murthly, Perthshire*,	1895
	Dr William Hartley Bunting, 22 *Islington Row, Edgbaston, Birmingham*,	1900
	Dr Francis Cadell,	1870
	Dr D. A. Carruthers, 5 *Charlotte Street, Perth*,	1901
	Dr S. W. Carruthers, 44 *Central Hill, Norwood, London, S.E.*,	1899
310	Dr R. D. Clarkson, *Falkirk*,	1896
	Professor Cleland, *The University, Glasgow*,	1864
	Dr A. R. Coldstream, 24 *Lung Arno Nuovo, Florence, Italy*,	1878
	Dr William Craig, *Cowdenbeath, Fife*,	1894
	Dr J. R. Crease, 2 *Ogle Terrace, South Shields*,	1885
315	Dr J. Frank Crombie, *North Berwick*,	1897
	Dr Bampylde Daniell, *Adderley Street, Cape Town*,	1903
	Dr P. M. Deas, *Wonford House, Exeter*,	1868
	Dr Frederick Dittmar, 72 *Murrayfield Gardens*,	1906
	Dr A. Home Douglas, 5 *Rue de Russie, Nice, France*,	1889
320	Dr J. J. Douglas, 42 *Central Hill, Upper Norwood, London*,	1891

		Date of Admission
	Dr C. C. Easterbrook, *Crichton House, Dumfries*,	1890
*	Dr Thomas Easton, 23 *East Park Terrace, Southampton*,	1894
	Dr J. W. Eastwood, 18 *Farndon Road, Oxford*,	1871
	Dr E. A. Elkins, *The Asylum, Leavesden, near Kings Langley*,	1895
325	Dr R. W. Felkin, 12 *Oxford Gardens, North Kensington, London, W.*,	1885
	Dr A. N. Fell, 11 *Devon Road, Colchester*,	1897
	Dr Samuel Ferguson, *Lyestone House, Ascot*,	1902
	Right Hon. Sir R. T. Finlay, Q.C., LL.D., *Hyde Park, London*,	1894
	Dr Charles Forsyth, *Alexandra Buildings, Hong Kong*,	1875
33	Dr Simson C. Fowler, *Juniper Green*,	1862
	Dr F. W. Lxcroft, 47 *North Street, Birmingham*,	1875
	Dr Dyce Fraser, 103 *Cornwall Road, London, S.W.*,	1885
	Professor Gamgee, *8 Avenue de la Gare, Lausanne, Switzerland*,	1895
	Dr William Gayton, 11 *Kelburn Avenue, North Finchley, London, N.*,	1886
335	Dr T. Paul Gray, 94 *Adelaide Road, Hampstead, N.W.*	1870
	David M. Greig, Esq., 25 *Tay Street, Perth*,	1872
	Dr W. C. Greig, *Otago, New Zealand*,	1884
	Dr George K. Grimmer, *Canada*,	1897
	Dr Groenbeck, *Cincinnati*,	1875
340	Dr W. B. T. Gulbin, *Redland Grove, Bristol*,	1893
	Dr John Haddon, *Denholm, Roxburghshire*,	1885
	Dr Archibald Hall, *Montreal*,	1853
	Professor D. J. Hamilton, *The University, Aberdeen*,	1876
	Dr J. W. Harrp, *Penn Road, Wolverhampton*,	1887
345	Dr John Hardie,	1895
	Dr A. W. Hare, *Newquay, Cornwall*,	1885
	Dr J. Home-Hay, *Alva*,	1880
	Professor J. Berry Haycraft, *Llandaff, Cardiff*,	1889
	Dr Stanley Haynes, *Malvern, Worcestershire*,	1894
350	Dr R. Douglas Helm, 13 *Portland Square, Carlisle*,	1882
	Dr David Hepburn, Professor of Materia, *University College, Cardiff*,	1902
	Dr R. L. Horsley, *Crux, Fife*,	1888
	Dr James Hunter, *St Catherine's, Lincoln*,	1900
	Dr W. Hunter, 103 *Harley Street, Cavendish Square, London, W.*,	1887
355	Dr Robert Inch, *Gorebridge*,	1887
	Dr W. Wotherspoon Ireland, 1 *Lauriston Terrace, Edinburgh*,	1883
	Dr D. W. Johnston, *P.O. Box 2022, Johannesburg, South Africa*,	1902
	Dr James Johnston, 53 *Princes Square, Bayswater, London, W.*,	1871
	Dr J. Carlyle Johnstone, *The Asylum, Morpeth*,	1882
360	Dr J. Keay, *Bangeur Asylum, Patna*,	1887
	Dr A. J. Keiller, *New Edinburgh, Carstairs*,	1880
	Dr George Keith, *Maidenhill, Carstairs, Carnwath*,	1845
	Dr Skene Keith, 58 *Upper Berkeley Street, Portman Square, London, W.*,	1885
	Dr W. Scott Lang,	1868
365	Dr J. W. Leech, 12 *Eldon Square, Newcastle-on-Tyne*,	1901
	Professor R. F. C. Leith, M.D., *The University, Birmingham*,	1888
	Dr John Linton, *Aberdeen*,	1863
	Dr G. R. Livingston, 47 *Castle Street, Dumfries*,	1901
	Dr Donald Macaulay, *Imperial Government Civil Service, Johannesburg, Johannesburg*,	1895
370	Dr John A. Macdougall, *Carstairs, Lanarkshire*,	1875
	Dr William C. M'Ewan, *Tredegar*,	1892

		Date of Admission.
	Professor Sir J. M'Fadyean, 101 *Great Russell Street, London, W.C.*,	1888
	Dr F. Reid Mackay, *New Zealand*,	1899
	Dr John Mackay, *Aberfeldy*,	1881
375	Dr W. B. Mackay, 23 *Castlegate, Berwick-on-Tweed*,	1893
	Professor M'Kendrick, *Maxie Burn, Stonehaven*,	1870
	Dr James Mackenzie, 68 *Bank Parade, Burnley, Lancashire*,	1894
	Dr Robert Mackenzie, *Napier, Nairn*,	1893
	Dr Theodore C. Mackenzie, *The Asylum, Inverness*,	1904
380	Dr William A. Mackintosh, 3 *Park Terrace, Stirling*,	1896
	Dr William MacKirdy, 157 *Lincoln Road, Peterborough*,	1907
	Dr David W. Maclagan, *care of Dr Harrison, Eltham, Taranaki, New Zealand*,	1901
	Dr J. Shaw M'Laren, *India*,	1887
	Dr Roderick M'Laren, 23 *Portland Square, Carlisle*,	1882
385	Dr William B. MacTier, 7 *Queen's Gardens, St Andrews*,	1901
	Dr Samuel Macvie, *Chirnside*,	1897
	Dr J. W. Martin, *Charterhall, Newbridge, Dumfries*,	1888
	Dr Andrew W. Messer, *Lemington, Scotswood-on-Tyne*,	1899
	Dr James Middlemass, *Boro' Asylum, Ryhope, Sunderland*,	1895
390	Dr E. A. Mills-Roberts, *Bethesda, Bangor*,	1897
	Dr J. Moolman, *Cape of Good Hope*,	1877
	Dr E. C. Moore,	1892
	Dr J. E. Moorhouse, 6 *Melville Terrace, Stirling*,	1901
	Dr A. E. Morison, *Mowbray Villa, Sunderland*,	1889
395	Dr J. Rutherford Morison, 14 *Saville Row, Newcastle-on-Tyne*,	1882
	Professor Robert Muir, *The University, Glasgow*,	1894
	Dr D. G. Macleod Munro, 1 *Oriel Villas, Cheltenham*,	1898
	Dr Peter Murray, *Durie Street, Leven*,	1904
	Dr W. H. Myers, *Indiana, U.S.A.*,	1906
400	Dr Leith Napier, *The General Hospital, Adelaide, South Australia*,	1879
	Dr Ernest F. Neve, *Srinagar, Kashmir, India*,	1884
	Dr Peter Orphoot, *Greenhythe, North Berwick*,	1865
	Dr T. Wyld Pairman, *Hinemoa, Moffat*,	1884
	Professor Bell Pettigrew, *St Andrews*,	1873
405	Dr Alexander Peyer, *Zürich*,	1893
	Dr J. A. Philip, *Rue Victor Hugo, Boulogne-sur-Mer, France*,	1878
	Dr J. H. Pringle, 172 *Bath Street, Glasgow*,	1886
	Dr S. Hale Puckle, *Bishop's Castle*,	1885
	Dr R. B. Purves, 5 *Minster Yard, Lincoln*,	1899
410	Dr J. Maxwell Ross, *St Ruth's, Maxwelltown, Dumfries*,	1882
	Dr J. R. Home Ross, *Burmah*,	1888
	Dr Joseph C. Ross, *Withington*,	1884
	Dr S. Rumboll, *Cavendish House, Leeds*,	1887
	Dr Thomas Russell, *Beechwood, Parkhead, Glasgow*,	1888
415	Dr Gordon Sanders, *Cannes, France*,	1892
	Dr F. A. Saunders, *Grahamstown, South Africa*,	1884
	Dr Thomas R. Scott, *Musselburgh*,	1884
	Dr W. Vernon Shaw, 42 *Yorkersgate, Malton, Yorks*,	1905
	Dr Herbert D. Shepherd, 45 *Kedleston Road, Derby*,	1903
420	Dr G. F. Shiels, 229 *George Street, San Francisco*,	1887
	Dr W. H. Shirreff, *Port Fairy, Victoria, Australia*,	1883
	Dr William Simmers, *Gordon, Berwickshire*,	1894
	Dr Herbert Sloman, 65 *Goldington Road, Bedford*,	1906
	Dr John Smith, *Brycehall, Kirkcaldy*,	1889
425	Dr William Smith, *America*,	1890
	Dr W. Ramsay Smith, *The General Hospital, Adelaide, South Australia*,	1892
	Dr Van Someren, *Northwood, Middlesex*,	1845
	Dr J. W. Somerville, 12 *Abbotsford Road, Galashiels*,	1905
	Dr A. M. Stalker, 156 *Perth Road, Dundee*,	1893

		Date of Admission.
430	Dr Graham Steell, 96 *Mount Street, Manchester*,	1877
	Professor Stephenson, *University, Aberdeen*,	1861
	Dr J. Lin Stevenson,	1900
	Dr Robert Stirling, 4 *Albert Place, Perth*,	1891
	Professor Ralph Stockman, *The University, Glasgow*,	1891
435	Dr H. K. Storer, *Newport, Rhode Island, U.S.A.*,	1855
	Dr John Struthers, *Ngamiland, Transvaal, South Africa*,	1895
	Dr J. F. Sturrock, *Arima, Bourbon, Jersey*,	1887
	Professor J. Symington, *Queen's College, Belfast*,	1878
	Dr C. C. Teacher, *North Berwick*,	1887
440	Dr C. Templeman, *Sanitary Office, Bell Street, Dundee*,	1891
	Dr J. A. Douglas Thompson, *Cornerton House, Haleswen, Manchester*,	1901
	Dr J. Stuart Thomson, *Castle Hill House, Dundee*,	1877
	Dr J. Batty Tuke, Jr., *Maryland*,	1886
	Dr R. S. Turner, *Keith*,	1867
445	Dr T. Edgar Underhill, *Dunedin, Mount Gould, Wolverhampton*,	1885
	Dr A. R. Urquhart, *Murray House, Perth*,	1893
	Dr Gopal Govind Vatve, *care of H.H. the Rajah of Miraj, Bombay, India*,	1895
	Dr Walter de Watteville, *Kingswear*,	1902
	Dr A. B. Whitton, *Aberdeen*,	1886
450	Dr H. Douglas Wilson, 3 *Kippen Road, Harrogate*,	1904
	Professor G. Sims Woodhead, *Pathological Laboratory, Museums, The University, Cambridge*,	1883
	Dr Yellowlees, 6 *Albert Gate, Knightsbridge, London*,	1862

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TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

FOR SESSION LXXXVI., 1907-8

Meeting I.—November 6, 1907

Dr J. O. Affleck, *President, in the Chair*

I. Election of Office-Bearers

The following gentlemen were elected office-bearers :—*President*, James Ritchie, M.D., F.R.C.S., Edin., F.R.C.P., Edin.; *Vice-Presidents*, Dr Byrom Bramwell, Dr George Hunter, and Dr Charles Watson MacGillivray ; *Council*, Professor Harvey Littlejohn, Dr G. Keppie Paterson, Dr William Guy, Dr Aitchison Roberston, Dr J. O. Affleck, Dr George A. Berry, Dr G. Lovell Gulland, and Dr Claude B. Ker ; *Treasurer*, Mr J. W. Dowden ; *Secretaries*, Mr David Wallace, C.M.G., and Dr Francis D. Boyd, C.M.G. ; *Editor of Transactions*, Dr William Craig.

II. Election of Members

Arthur John Brock, M.D., Ch.B., 27 Braid Crescent ; James Burnet, M.D., M.R.C.P., Edin., 6 Glengyle Terrace ; Bernard Ramsay Craig Christie, M.D., 27 Inverleith Gardens ; William MacKirdy, L.R.C.P., Edin., 157 Lincoln Road, Peterborough ; and James B. Mears, L.R.C.P. and S., Edin., L.F.P.S., Glasgow, 36 Nile Grove, were elected Ordinary Members of the Society.

III. Treasurer's Annual Statement

EDINBURGH MEDICO-CHIRURGICAL SOCIETY'S ACCOUNTS

FOR SESSION 1907-8.

INCOME.				EXPENDITURE.				
			£167 12 8					£163 10 7
Entrance Fees	£14	14	0	Turnbull & Spears, printing Vol. XXV. of Transactions	£95	17	11	
Annual Subscriptions	128	10	0	J. M'Callum, Tea and Coffee	6	4		
Annual Subscriptions, arrears	0	10	0	A. Frazer, Microscopes, Lantern	10	19	6	
Annual Subscriptions in advance for 1907-8	0	10	0	Collector	3	10	0	
Subscriptions for Transactions	0	10	0	Doorkeeper	1	6	0	
Dividends on Caledonian Railway Stock	13	0	0	Illustration of Papers—				
Interest on Deposit Receipt	9	12	8	Dr Sinclair	5	5	0	
	£107	12	8	Dr Sherman	1	3	6	
				Dr Boyd	1	8	2	
				Rent of Hall	10	0	0	
				Neill & Co., Billets, Postage	26	1	6	
				Dr G. Lovell Gulland, Petty Cash	1	0	0	
				Petty Cash	0	15	0	
					£163	10	7	
Balance in Bank, October 1906			70 4 6	Balance in Bank			33 16 7	
				Paid to Deposit Receipt			40 0 0	
				Cash in Hand			0 10 0	
			£237 17 2				£237 17 2	

STATE OF FUNDS.

At November 1906.				At November 1907.			
On Deposit Receipt	£260	0	0	On Deposit Receipt	£300	0	0
Caledonian Railway Debenture Stock (Purchase Price)	484	11	6	Caledonian Railway Debenture Stock (Purchase Price)	484	11	6
Balance in Bank	70	4	6	Balance in Bank	33	16	7
	£814	16	0		£818	8	1

J. W. HOWDEN,
Treasurer.

IV. Exhibition of Patients

1. *Mr J. M. Cotterill* exhibited—

(*a*) Male, aged 35. Some eighteen months ago the patient had a fall from a dogcart, striking the back of the head and becoming unconscious for two hours. The next day, proptosis of the right eye was noticed, with diplopia and loud buzzing in the head, preventing sleep.

An angioma developed in connection with the ophthalmic branch of the internal carotid artery; and free anastomotic connection could be recognised in the angular branches of the facial artery on both sides. The right common carotid artery was tied, and the condition has been entirely cured.

(*b*) Male, aged 60, with enormous LYMPHOSARCOMA of the right posterior triangle of the neck, axilla, and anterior chest wall. The case being inoperable, it was suggested to try the effect of X-rays.

2. *Mr Alexis Thomson* exhibited—

(*a*) A case of PATHOLOGICAL FRACTURE OF THE LOWER END OF THE FEMUR. The patient, a boy aged 9, had been sent into the Deaconess Hospital by Dr Watt of Falkirk, with the history that for some months he had suffered from pains above the left knee, and these were regarded as of the nature of growing pains. While standing in the street the left leg gave way under him, and he fell to the ground; and it was found that he had sustained a fracture at the junction of the middle and lower thirds of the shaft of the left femur, and on examination under chloroform the bone was felt to be enlarged, and the fragments could be moved on one another without coarse grating. The other bones of the skeleton appeared to be normal. An X-ray photograph showed that the architecture of the lower third of the diaphysis of the femur was greatly altered, that there was considerable rarefaction both of the cortex and of the internal spongy bone, and in the latter there were rounded areas which might correspond to cysts. The bone was sensitive on pressure. Satisfactory union took place at the end of eight weeks. The further progress of the case showed it to be one of sarcoma of the femur; the limb was amputated below the lesser trochanter, and the patient made a good recovery.

(*b*) An UNUNITED FRACTURE OF BOTH BONES OF THE FOREARM. The patient, a boy aged 9, was admitted to the Deaconess Hospital on the recommendation of Dr Murray of Leith. The history was

to the effect that two years ago he had fallen on the hand and sustained a fracture of both bones of the forearm. Some months later he came under the care of Dr Adam of Dingwall, who found that the bones of the forearm were bent at a right angle, and who performed an operation with the object of straightening the bones, with an apparently satisfactory result. He was then lost sight of, and when seen again it was found that the angular bend in the forearm had been reproduced, and at the present time the fragments can be moved upon one another. Mr Thomson proposed to operate with a view to correct the deformity and fix the bones.

3. *Mr H. J. Stiles* exhibited

(*a*) A woman, aged 70, after excision of the upper jaw (along with the malar bone and floor of the orbit) for a LARGE ROUND-CELLED SARCOMA, which had commenced in the region of the antrum. The tumour had perforated the antrum, and involved the deeper tissues of the cheek. The patient, although very advanced in years, was otherwise healthy. Considering the serious nature of the disease and the distressing outlook if it were left to nature, it was thought right to give her the chance of an operation.

The first step in the operation consisted in introducing a laryngotomy tube through the thyro-hyoid space, and then plugging the lower part of the pharynx with a sponge. The anæsthesia was then administered through the laryngotomy tube. After excising the jaw and the malar bone, the posterior ethmoidal and sphenoidal sinuses were found to be full of muco-pus, and were therefore opened up and disinfected. After carefully suturing the flap, the cavity was stuffed with gauze rung out of turpentine. Mr Stiles remarked that he was very fond of turpentine in these cases, as it was hæmostatic as well as strongly antiseptic and, being volatile, it lessened the risk of septic pneumonia. There was no shock to speak of, and the patient made a rapid recovery.

Mr Stiles said he was formerly in the habit of performing a preliminary tracheotomy in such cases, but recently he had substituted laryngotomy, which he found simpler and equally efficacious. The great advantage of being able to plug the pharynx was that there was no fear of blood getting into the lungs, and the whole operation could be done quietly and much more accurately and deliberately.

(*b*) A patient after repeated operations for EPITHELIOMA OF THE FLOOR OF THE MOUTH. The patient first came under treatment

in July 1905, for a small epithelioma of the floor of the mouth, about the level of the canine tooth. The tongue was not implicated, but as the tumour approached very near the gum a portion of the body of the jaw was removed as well as the glands in the sub-maxillary region.

In January 1906, the patient returned with recurrence in the floor of the mouth and under surface of the tongue. The glands in both sub-maxillary regions were involved, as well as those in the carotid triangle on the affected side. A preliminary tracheotomy was performed, and the whole of the tongue, the floor of the mouth, the body of the jaw, and the sub-maxillary glands on both sides were removed by a curved incision in the sub-maxillary region, extending from a little below the angle of the jaw to a similar point on the opposite side. The glands in the carotid triangle on the left side were removed by an incision carried downwards along the anterior border of the sterno-mastoid.

In January 1907, the patient again presented himself with a large malignant glandular tumour growing from the upper part of the left anterior triangle of the neck. The skin covering the swelling was reddened, and the subjacent glands were beginning to break down. There was no recurrence inside the mouth, and as the patient was very anxious that Mr Stiles should attempt to remove the disease, he acceded to his request. After removing the glands and the skin covering them, the gap, which extended from the chin above to the thyroid cartilage below, and from one sterno-mastoid to the other, was almost entirely closed by splitting the lower lip in the middle line and turning the two flaps downwards, and suturing them to the edges of the wound in the neck. In this way the mouth and lower part of the pharynx presented one continuous cavity at the bottom of which the epiglottis was seen to project upwards. After the parts had healed, one could obtain a very striking demonstration of the behaviour of the epiglottis during the act of swallowing, and of the manner in which the vestibule of the larynx was closed. What happened served conclusively to prove the error of the old idea of the epiglottis being folded backwards over the vestibule, and to bear out, in every detail, the accuracy of the mechanism described by Professor Anderson Stewart. As stated by him, the first step consists in the obliteration of the posterior part of the rima by the approximation of the arytenoid cartilages, and then the closure of the glottis is completed by these being drawn forwards so as to come

in contact with the cushion of the epiglottis, the latter remaining stationary.

The patient presented himself in June 1907, with recurrence in the neck involving the skin in the hyoid region as well as the deep cervical glands along both internal jugulars. In spite of this recurrence the patient had gained 27 lbs. since the last operation. Mr Stiles pointed out to him that he was afraid anything further in the way of operation would be useless. He, however, insisted that Mr Stiles should make another attempt to remove the disease. The patient had always displayed such courage and fortitude at the former operations, that he felt he could not refuse him this last chance. He accordingly again opened the trachea, and, after plugging the pharynx, removed the hyoid bone along with the involved skin, and a chain of malignant glands along each internal jugular, including two or three inches of the vein itself on the right side. The gap in the neck was almost entirely filled in by first freeing the mucous membrane from the anterior aspect of the epiglottis, and then folding it (along with the separated mucous membrane) downwards, and stitching it to the skin edges bounding the gap in the neck. After the wounds had healed, the vestibule of the larynx was fully exposed, and it was again interesting to note how the rima glottidis was closed in the manner above mentioned, even after the epiglottis had been folded downwards to cover in the raw surfaces on the front of the neck.

Mr Stiles remarked that he had been deeply touched by the bravery and fortitude which the patient had never ceased to display since the beginning of his illness, and he was greatly grieved to have to confess that the disease had again recurred in the glands in the neck, and that further operation was quite out of the question.

(c) A male, aged 21, after NEPHRO-LITHOTOMY. The interest of this case lay in the fact that the patient had suffered many years ago from tubercular disease in the dorso-lumbar region, complicated with a psoas abscess. For the past six years he had complained of more or less persistent pain in the loin on the same side as the abscess. The pain did not radiate along the genito-crural nerve, nor was there ever any hæmaturia. There was tenderness on deep pressure over the kidney, and the urine contained a little pus and some oxylates, but no tubercle bacilli. The diagnosis lay between tuberculous disease of the kidney and renal calculus. An excellent X ray photograph, taken by Dr Edmund Price, showed a slightly irregular calculus the size of the terminal phalanx of the

thumb, situated just external to the tip of the transverse process of the second lumbar vertebra.

The interest of the operation lay in the fact that the old spinal disease had so diminished the ilio-costal space that access to the kidney was very difficult. Fortunately, however, the stone could be felt in the pelvis of the kidney, and by scraping through its wall with the finger nail, the calculus escaped, and was then extracted from the bottom of the wound by a pair of long dressing-forceps. No urine escaped from the wound after the tenth day, and at the end of the third week the patient was able to get up with the wound quite healed.

V. Exhibition of Specimens

Mr J. M. Cotterill exhibited—

(*a*) Uterus and appendages from a case of panhysterectomy in a woman of 42, suffering from fibroids of the uterus and ovarian dermoids. There was considerable pain in the abdomen and difficulty in defæcation. The patient made an excellent recovery.

(*b*) Pyosalpinx complicating appendicitis. The pyosalpinx was apparently the original source of trouble, and the appendix had become adherent to the tube, dragged out of position, and kinked. Recovery.

(*c*) Hydatids of the liver from a man aged 38, who had been infected by his own fox terrier. There were two separate cysts, one at the free margin of the liver, and one at the apex. Recovery.

(*d*) Sarcoma of the foot from a female aged 23. Amputation. Recovery.

(*e*) Tumour of the cerebellum (sarcoma) successfully removed from a male patient aged 34.

(*f*) Tumour of the cerebello-pontine angle removed p. m. from a male patient aged 48. "Decompression" had been performed with temporary relief.

VI. Original Communications

1. NATURE'S OBJECT-LESSONS IN DISEASE

PRESIDENT'S VALEDICTORY ADDRESS

By J. O. Affleck, M.D., F.R.C.S.Ed., F.R.C.P.Ed., Consulting Physician, Royal Infirmary

Gentlemen,—My predecessor in the Presidential Chair of this Society, when delivering his Valedictory Address two years ago, expressly refrained from merely taking a survey of the work done

during his tenure of office; and in choosing for a subject between a distant look backward and a distant look forward, selected the former, and gave a highly interesting and memorable historical sketch of the Society and some of its more prominent members during the previous fifty years.

I do not think that there ought to be any standing pattern for such addresses, although there will ever be some points of reference more or less common to all. In the parting words which constitute this Valedictory Address I desire to confine myself to a look rather at the present than at the past or the future.

It will be in the recollection of some at least of the members of this Society that on the first occasion of my occupying the chair I gave a cordial invitation to the younger members to take a part in the work and the discussions, pointing out the educative value this possessed for them. It is a gratifying fact to me on this, my last appearance as President, to recall the response which this invitation received, for there have been few meetings in which important contributions, either in the form of separate communications or of sharing in the discussions, have not been made by those who enjoy the enviable privilege of being numbered among our younger members.

May I venture once more to renew the appeal? The future of a Society like this, as a centre of scientific influence in our profession, lies largely in the hands of the young.

So much am I impressed with this that in the few words I speak to-night I desire to have particularly in view the younger members of the Society. They will, I feel sure, acquit me of any desire to assume the attitude of lecturing them. On the contrary, there is much I have learnt, and am daily learning, from contact with the well instructed, fresh-thinking and enthusiastic minds of my younger brethren. But is it too much to hope that they on their part may find food for thought in the reflections of one who, throughout more than a generation, has been as he still is, in daily touch with disease and its manifold problems, has served in various responsible capacities of professional life, has been privileged to take a share in the teaching of students, and who continues to maintain an interest in all that relates to the advancement of medicine, which will, he hopes, only cease with his life? With such privileges and opportunities it could scarcely fail that impressions should be formed and reflections arise as to the position of the medical science of the present time, and the trend of its

progress. These must of necessity be largely from the point of view of the physician. There is nothing more conspicuous in the period covered by that to which I have referred than the development of the scientific spirit among workers in every department. Let any one look into the current medical literature of twenty-five or thirty years ago, and compare it with that of the present day, and say whether in many respects we are not now reading a new language—a language born of research into the occult processes of disease. The relation of micro-organisms to morbid action has opened up, in the science of bacteriology, a new field of inquiry, and produced a new literature. This has broadened the scope of medical science, and made possible other lines of investigation of the most far-reaching importance. It would be beyond the purpose of this address to enlarge upon this topic, but one or two points may be referred to. In regard to some of the infective processes it is not so long since the discovery of specific micro-organisms was itself held to be a great stride towards their true pathology. But this was felt to be at best a limited gain, so long as we were without knowledge of the products of these parasites, and of the soil conditions which made their growth and lethal action effective. Thanks to the labours of the army of workers on the subject of immunity, much light has been shed upon the properties of serums, and their action upon bacteria and their toxins, with results in the case of some maladies that justly take rank among the therapeutic triumphs of our age ; while there still seems to be a limitless field for investigation in this domain. The minute examination of the blood, as now practised and taught, has been of immense service in helping the solution of many diagnostic problems alike in medicine and surgery. Clinical chemistry as applied to the processes of digestion, and the investigation of various secretions, has been of diagnostic service, and has given promise of a hopeful future for this hitherto little cultivated branch of science. In like manner the application of physics, as, for example, in the estimation of blood-pressure, in the various uses of electricity, and in the methods of radiography has contributed in no small measure to the accurate investigation as well as to the treatment of many morbid states. These are but a few illustrations of the advance medicine is making along scientific lines towards the discovery of truth.

These subjects now enter largely into the clinical teaching of the present day, and all who are engaged as instructors of

students, as well as all who seek the advancement of medicine, must hail with satisfaction every step forward that science makes possible.

Nevertheless there will arise from time to time in the minds of not a few the question whether, in the large share of attention that these scientific methods claim and receive, there may not be the danger of overlooking altogether, or failing to give due consideration to, other sources of information not less important, and certainly not less interesting, if searched for and observed.

I. Nature's Object-Lessons in Disease

That nature responds to morbid processes in the body in a manner more or less obvious to the senses is a truism that all will readily admit. But it is doubtful whether we habitually avail ourselves of all the information capable of being obtained by an attentive observation and study of altered appearances and characteristics. This information may in some instances be direct and accurate, in others indirect and suggestive. Accordingly, a consideration of the physiognomy of disease seems to possess a legitimate claim upon our attention. This word physiognomy is not meant to apply merely to the *facies* of disease, but to all the changes capable of being appreciated by our unaided senses. But, even taking the more restricted significance of the term, is it not the case that as regards not a few diseases nature has depicted them upon the countenance? Not only may this be seen in many of the diseases of the blood where the aspect is unmistakable, but much may be made out, both as regards the identification and the progress of the case, from a study of the *facies* in such maladies as pneumonia, phthisis, Bright's disease, various forms of heart disease, chronic gastric disorders, exophthalmic goitre, myxœdema, general paralysis, and many others. Instances manifold arise in one's memory of helps to diagnosis obtained in this way. One or two taken almost at random might be quoted. (*a*) A young clergyman comes complaining of constant dyspeptic troubles, with headache, vomiting, and growing weakness, which the many stomachic remedies he has received have failed to remove. As he narrates his symptoms the physician scrutinises the patient's countenance and notices a peculiar glistening appearance of the eye (the dropsical conjunctival) which immediately suggests an examination of the urine, when the nature of the ailment is revealed for the first time. (*b*) A middle-aged man comes for examination for life

assurance. He declares he has never been ill in his life. The medical examiner, who had never seen him before, observes a certain dryness and thinness of the skin, and a general dull flush of the face, and the suggestion of diabetes comes into his mind. The urine is examined and is found to contain a large quantity of sugar. (*c*) A case of prolonged and persistent high temperature in a young woman in the Royal Infirmary, which for a time caused great difficulty from the otherwise negative symptoms, presented in her countenance such an aspect of well-being as to suggest that this might be one of those rare examples of hysterical pyrexia occasionally met with—this diagnosis being amply confirmed by the collateral evidence and by the subsequent progress of the case.

The *facies* is of wonderful assistance in the diagnosis of typhoid fever, and during my long association with my friend Dr Ker in the work of the City Hospital I have invariably observed that the first note he makes on the case of any newly admitted patient is as to whether he or she looks or does not look like the disease.

My surgical brethren are only too familiar with the abdominal *facies* and its significance in view of, or subsequent to, the operation of laparotomy. Lastly, it seems hardly necessary to recall the remarkable change in the countenance observed on a favourable crisis in such diseases as pneumonia and typhus fever.

Apart, however, from facial evidences, there are numerous other objective indications which may prove largely contributory in determining a diagnosis. The attitude, the gait, the voice, the speech, the play of the features, the characters of a cough, and many other phenomena may be notable sources of information.

The first of the seven cases of beri-beri I have in the course of my experience had opportunity of seeing and treating was revealed to me in a rather singular manner. A young sailor just returned from South America was sent to me at the Royal Infirmary as a case of probable malaria. His sallow aspect was certainly suggestive of this, but as he stood, and particularly as he walked, it was obvious that some other ailment existed from the manifest weakness of his lower limbs. At the moment there happened to be with me two medical friends from China, and on my calling their attention to this patient one of them said to the other, "That looks very like the walk of beri-beri." The man instantly replied, "That was what they called my trouble when I was in the hospital in Brazil." The case proved to be a typical example of the disease.

It has long seemed to me that much might be learnt from the study of the character of coughs. I have the belief that in every disease which has cough for one of its symptoms there may be found something more or less characteristic in the cough. Certainly there are some which admittedly have their individual characters, such as the short suppressed cough of pneumonia, the irritable dry cough of early phthisis, the hysterical cough, the barking cough of puberty, the cough of chronic bronchitis in old people, the cough of capillary bronchitis in children, to say nothing of the spasmodic cough of whooping-cough, the clanging cough of intra-thoracic pressure, and many others. I have sometimes fancied the cough in a case of pyopneumothorax had a sound of its own.

It is hardly necessary to recall the fact that but for objective indications it would many a time be practically impossible to recognise and interpret the evidences of disease in children.

These various examples are merely mentioned to illustrate the point I wish to emphasise, namely, the value of the information that so often lies ready to hand, and that is too often overlooked.

But certain difficulties may be suggested —

1. May not such information be apt to lead astray? Doubtless, were it to be relied on alone; and more cases might be chronicled from one's experience of failure than of success in attempting to recognise disease from external manifestations only. But no one ever trusts in diagnosis to one line of evidence when many are available.

2. What then can be regarded as the real value of such methods, seeing they are confessedly uncertain? Their value lies in the aid they afford to the prosecution of further inquiry, also in the suggestions and associations of ideas which are awakened in the mind when the attention is directed to particular points in an investigation.

3. Granting all this, do these so-called object-lessons in disease not more properly belong to the period in professional life when sufficient experience has been gathered to utilise them? This is only partly true. These phenomena are open to observation, and the exercise of this faculty, which lies at the foundation of all clinical progress, cannot be begun too early. Assuredly it may be cultivated by all notwithstanding the fact that it comes easier to some than to others. It is at no time an entirely easy matter, seeing that it includes a great deal more than mere per-

ception. In an interesting address delivered some years ago by that distinguished surgeon, the late Sir James Paget, he refers to the "power of careful observation and cautious thinking," and adds: "In ordinary talking men seem to think that careful observation is one of the easiest things in the world; and there are some who speak as if they themselves had never overlooked anything of importance. Study the history of any science and you can see evidence of the difficulty of observation on every page. It would be a very useful book, I think, if some one would write, not a history of discoveries, but a history of oversights."

Now it surely cannot be thought that the limits of observation here advocated could ever be hostile to, far less displace, the most sedulous prosecution of the methods of scientific investigation which are now in such abundance placed within our reach. On the contrary, they but play a part, yet a necessary part, in any system of inquiry which could be regarded as at all complete. Diagnosis is in too many instances a difficult and uncertain conclusion—a summation of probabilities, and no aid thereto can safely be omitted.

It has been affirmed of some eminent literary characters that they possessed the power of being able to find out all that was in a book by merely glancing over a few pages here and there. The book of disease, as set forth in the individual, is not to be read to any good purpose in this fashion; but every page, every sentence, every word must be studied and weighed if we are truly to interpret its contents. As one who has, over a long series of years up to the present time, enjoyed the privilege of engaging in clinical teaching, might I venture, with all respect, to suggest to my brethren in the work the importance of keeping before the student the fact that nature has object-lessons to teach regarding many—perhaps nearly all—diseases, if only we can find them out and read them?

II. Clinical Impressions

The careful prosecution and the mental registration of observations in disease lead, in process of time, to the experience of what, for want of a better term, I would venture to call clinical impressions. By that is meant the view that is conveyed to the mind, when brought in contact with any case of disease, as to the patient's condition and his prospects. This, whatever may be thought of it, has long appeared to me to be one of the most valuable

assets in the practitioner's possession, which, moreover, may oftentimes be turned to practical account in his efforts at treatment. For confirmation of this my appeal must of course be made mostly to my senior brethren. Is it not the case that in not a few instances there comes to one an impression or an admonition, which could not find expression in words, but which yet gives a view of the patient's state, that often proves to be true, and which may indicate at the same time a line of treatment that might otherwise have been overlooked? One advantage which this acquisition possesses consists in the power it sometimes gives us of inspiring hope in circumstances where appearances might indicate the contrary. No doubt such impressions as I have referred to may occasionally be ill-founded or erroneous, and certainly they are never to be put in competition with, still less made to substitute, the most detailed and exhaustive examination possible, but yet that there is on the whole a substantial practical value in them cannot, I think, be seriously disputed.

III. The Psychical Factor and its Relation to the Practitioner

It might seem at first sight that any allusion to this subject was somewhat inconsequent upon an address devoted mainly to the objective manifestations of disease. But this is far from being the case, since this element can never be wisely excluded by either physician or surgeon. It is never to be forgotten that when called to deal with any case of illness we are dealing not merely with pathological phenomena, but with the man, or woman, or child who is the subject of them. And just as certainly as that few diseases are not attended with some more or less observable outward and visible sign, so there are few where the mental or psychical elements of the nature are left wholly untouched, and their influence and reaction upon each other form oftentimes an important and difficult problem. Sometimes the mental outweighs the physical, and has to be reckoned with and treated before the health can be restored. But probably more often the reverse holds good.[1]

[1] One is here reminded of the story told by the great English physician, Sydenham. He had been long attending a gentleman of fortune with little or no advantage, when at last he frankly avowed his inability to render him any further service, adding at the same time that there was a physician of the name of Radcliffe at Inverness who had distinguished himself by the performance of many remarkable cures of the same complaint as that under which his patient laboured, and expressing

Cognisance must be taken of these things as they affect both patient and practitioner. It is as interesting as it is striking to observe and study the mental attitude of a patient labouring under some severe or long-standing illness. In some happy instances a change of the most salutary and benign character presents itself, and the qualities of fortitude, patience, cheerfulness, and hope (together with a certain effort to live or to be well) shine out conspicuously even in natures which were of such construction as would never have justified the anticipation. The favourable influence of such mental states upon the course and progress of many forms of disease is real and unmistakable. In other instances, impatience, irritability, opposition, and despondency are painfully and disappointingly apparent, and add gravely to the burden of the sufferer, while seriously prejudicing the course of the disease. Of all these latter-named mental complications, despondency or depression is that which justly causes most concern, for it can hardly be doubted that this may sometimes be the one and only thing that stands between the patient and his recovery. There arise in my memory more than one instance in which the sufferer from the first pronounced his own sentence and proceeded to die when the physical conditions, as far as could be ascertained, by no means justified so untoward an issue.

Facts of this kind lay upon the practitioner the necessity and the duty of taking account of the mental position of the patient, just as much as of those external manifestations or object-lessons for the importance of which I have been contending. They are both the product of disease, and neither of them can be excluded from any true estimate of the case.

This reckoning with the patient's mental condition, alike for

a conviction that if he applied to him he would come back cured. This was too encouraging a proposal to be rejected; the gentleman received from Sydenham a statement of his case, with necessary letter of introduction, and proceeded without delay to the place in question (about two and a half centuries ago). On arriving at Inverness and anxiously inquiring for the residence of Dr Robertson he found to his utter dismay and disappointment that there was no physician of that name, nor had been in the memory of any person there. The gentleman returned vowing eternal hostility to the peace of Sydenham, and on his arrival at home instantly expressed his indignation at having been sent on a journey of so many hundred miles for no purpose. "Well," says Sydenham, "are you in better health?" "Yes! I am now quite well, but no thanks to you." "No," says Sydenham, "but you may thank Dr Robertson for curing you. I wished to send you a journey with some object of interest in view: in going you had Dr Robertson and his wonderful cures in contemplation: and in returning you were equally engaged in thinking of scolding me!"

diagnosis and treatment, may impose demands upon the forbearance and charity of the practitioner, but the output of these virtues will in the long-run seldom be in vain.

I have directed attention to the importance of the scrutiny of the face in disease. But it is ever to be remembered that there is another aspect of the matter, and that is the scrutiny of the practitioner's face by the patient. It is surprising with what penetration and accuracy some sufferers will read into the thoughts and views, the hopes or fears, of their medical attendants. It is well if the patient can read in the physician's face that which inspires confidence, and brings hope and courage. It is better if, in addition, he recognises that divine quality of sympathy which seems to lighten and bear away some of the heavy burden of sickness, and gives strength and solace even in the presence of the last enemy.

Gentlemen, it only now remains for me to ask you to accept my cordial thanks for the honour you did me in electing me to occupy the chair of this Society two years ago, and especially for the uniform kindness, courtesy, forbearance, and support I have received during my tenure of office, which have made the position not only an easy but also an extremely pleasant one.

It has been truly written, "Better is the end of a thing than the beginning thereof." But, viewing the matter from the personal standpoint, and without affectation, it seems that in my case the sacred axiom might have to be read in the reverse way, since I fear the promise appeared better than the performance has proved. I had hoped to do more, and certainly no one can be associated with the affairs of this Society without becoming aware that it is in many directions still open to improvement—one serious obstacle being the want of premises we can call our own. As to the work done in the Society during these two past years, it may be sufficient to say that if there has been nothing very outstanding it has yet been of good, solid, and informing quality—work of which the Society has no reason to be ashamed. Speaking for myself, I can say that during the many long years I have been a member I have never attended a meeting without coming away richer in facts than when I entered the room, and the same is true while I have been occupying the Chair. The Society continues to grow and flourish. The attendance and the manifest interest shown have been a source of gratification.

And now, in resigning the duties of the Chair into the capable

hands of my successor, I would express my earnest wishes for the continued prosperity of the Society and its work, and assure the members that my interest in it will be not the less, but all the greater, from my having been privileged to hold the honourable position of its President.

2. PRELIMINARY NOTE ON A SIMPLE OPERATION FOR UNCOMPLICATED OBLIQUE INGUINAL HERNIA IN YOUNG ADULTS.

By George L. Chiene, M.B., F.R.C.S.Ed., Assistant Surgeon, Edinburgh Royal Infirmary

"The natural power of recovery is often so great that all that is needed is but to remove any unfavourable condition."—Mackenzie (Burnley), *Caledonian Medical Journal*, July 1907.

Mr President and Gentlemen,—Last month on mentioning the subject of this paper to a physician he remarked, "I thought you surgeons had said all there is to say about hernia"; yet Rigby (1), in his review of recent literature on hernia, commences with the following sentence: "It seems extraordinary that a definite decision has not yet been arrived at as to the origin of the hernial sac."

The *rationale* of the operation which I wish to bring to your notice this evening depends upon the following hypotheses:

1. That for practical surgical purposes all oblique inguinal hernias are primarily due to a congenital or preformed sac.

2. That in young adults suffering from oblique inguinal hernia the muscles guarding the internal abdominal ring will regain their normal valvular action if the cause of the dilatation of the canal be removed.

3. That if the neck of the sac be efficiently dealt with, it is quite unnecessary to interfere with the remaining portion in the inguinal canal or scrotum.

Two years ago I made various dissections of the inguinal region in the cadaver, and, as a result, devised a simple operation, the steps of which are the following:—

Operation

An incision is made half an inch above and parallel to the *middle* third of Poupart's ligament, the centre of which corresponds to

the position of the internal abdominal ring. This incision exposes the external oblique muscle, which is incised in the direction of

FIG. 1.—Shows relation of incision to Poupart's ligament. A. The middle of Poupart's ligament.

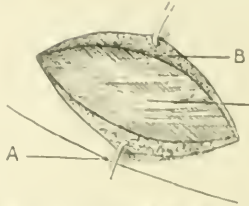

FIG. 2.—A. The middle of Poupart's ligament. B, Retracted skin, fat, and fascia. C, external oblique muscle.

FIGS. 2, 3, 4, and 5 (actual size) show different stages of the operation.

its fibres and retracted, thus exposing the lower portion of the internal oblique muscle and the upper portion of the cremasteric. The fibres of the latter are divided in the same direction as those

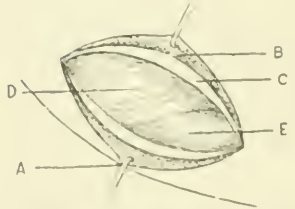

FIG. 3.—A and B as above. C, external oblique muscle retracted to [illegible] of internal oblique muscle. E, fibres of cremasteric muscle. (The ilio-inguinal nerve is not represented in the drawing.)

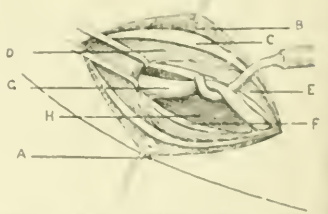

FIG. 4.—A, B, and C, as above. D, internal oblique muscle retracted upwards, after cremasteric muscle has been split. E, split cremasteric muscle. F, spermatic cord. G, neck of sac freed from cord. H, position of cord which is not represented.

of the external oblique muscle. The lower edges of the internal oblique and transversalis muscles are then retracted upwards and outwards, and the transversalis fascia exposed. This fascia is dealt with in the same manner as the external oblique and cre-

masteric muscles. The neck of the sac is thus exposed at the *internal* ring. It is freed from the cord, opened, and divided. The neck is ligatured and fixed to the under surface of the abdominal wall, above and external to the internal abdominal ring, the rest of the freed portion being cut off. This concludes what is sometimes termed the " first stage " of the operation.

The second stage consists in suturing the split fibres of the external oblique by the overlapping method, and closing the skin incision. In some cases the fibres of the cremasteric muscle are drawn together with a single stitch, but in the majority they will fall into apposition naturally.

It will be noted that the internal oblique and transversalis muscles are left untouched, also that the sac, with the exception of a small portion at the internal ring, is left *in situ*.

The reasons for my belief in the hypotheses on which the operation is founded are as follows.

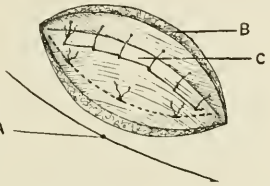

FIG. 5.—A, B, and C as above, showing the overlapping of the divided external oblique muscle. The dotted line shows the lower edge of the upper fibres drawn under the lower fibres and retained by three interrupted mattress sutures, the upper edge of the lower fibres fixed by a continuous blanket suture.

ANATOMY OF HERNIA

With regard to the first—that for practical surgical purposes all oblique inguinal hernias are primarily due to a congenital or preformed sac—it is necessary that we understand exactly what is meant by the word " congenital " when used in relation to inguinal hernia. Originally the term was confined to that variety of hernia in which the sac was in direct communication with the tunica vaginalis ; modern textbooks, however, include under this heading not only the variety mentioned, but also the varieties termed funicular, infantile, encysted, and interstitial. Any one who has had the opportunity of operating on a fairly large number of inguinal hernias in children will have recognised that only a minority of the cases dealt with come under the heading " congenital " as originally defined.

Stiles (2), in a consecutive series of 360 cases in children, found that in only 5 per cent. did the sac communicate with the tunica

vaginalis; other writers on inguinal hernia in children have noted similar results. Russell (3) maintains that the term "congenital," which he says, "has been the source of endless obscurity and confusion in the past," should be confined to those cases in which the hernia is present at birth. Without further elaboration of this point, it will be sufficient to say that I believe all oblique inguinal hernias to be due to a preformed sac, whether partial or complete.

As already indicated, most authorities now admit this to be the case in children. What proof is there that it is not so in adults also? The textbooks divide oblique inguinal hernias into congenital and acquired, but when we search for a definite statement which will lead us to recognise the acquired variety we are grievously disappointed. Murray (4) says he has been quite unable to distinguish between the congenital and the so-called acquired inguinal sac, for the very good reason that, in his opinion, they are one and the same thing.

Russell (3) for eight years has advocated what he now terms the "saccular" theory of hernia, and has stated his opinion that this theory will be either universally accepted or universally rejected in the near future.

Stiles (2) in 1904 wrote: "It is more than probable therefore, that the majority of oblique inguinal hernias which are met with during adolescence and in young adults are essentially congenital."

Recent papers by other authors (Bull and Coley (5), Clogg (6)) admit the probability of a preformed sac in adults.

Keith opposes Russell's theory, but brings forward no evidence to show that the preformed sac is not the essential cause of that variety of hernia we are at present discussing. Murray (4), on the other hand, produces positive evidence in support of the saccular theory. Lockwood (8) and Langton (9) draw attention to the fact that in many instances a preformed sac is present without there being any evidence of hernia.

The condition found in a patient, 30 years of age, on whom I operated is of interest in this connection.

The operation just described was performed for an oblique inguinal hernia on the right side. Owing to the laxness of the canal on the left side the internal ring was cut down upon, although no signs of hernia had ever been present. A patent tube of peritoneum about an inch long was exposed and removed. Lockwood (8) mentions a similar case.

An inguinal hernia which develops on the opposite side to that

already operated on is probably due to a condition similar to the above.

Some surgeons believe that if a hernia in a young adult develops suddenly that is proof that there is a preformed sac; but if, on the contrary, the hernia manifests itself slowly, it is acquired.

This second deduction is, in my opinion, unwarranted. There is no reason why a hernia should not appear by degrees, even although there is a preformed sac. It is just as likely that the slow development of the hernia is due to the fact that the contents have to dilate the preformed sac, whose lumen may be small, as that the contents have to push a process of peritoneum in front of them, thereby forming a sac.

If we look back we find that discussions in the past centred round the question of rupture *versus* dilatation of the peritoneum. When the former was finally disproved, all interested turned their attention to the possible causes of dilatation, taking it for granted that, because rupture was disproved, therefore the alternative—dilatation—was correct. By no means a sound deduction.

If the "saccular" theory be admitted with regard to oblique inguinal hernia, the same principle which guides us in the operative treatment in children holds good in adults.

The second hypothesis is that in young adults, suffering from oblique inguinal hernia, the muscles guarding the internal abdominal ring will regain their normal valvular action if the cause of the dilatation be removed.

It will, I think, be admitted that the weakness of the muscles in the region of the inguinal canal must be due to one of two causes —either a delay in their development or a weakening of normal muscles due to prolonged and increasing dilatation of the canal by the contents of the sac.

With regard to the former, writers on inguinal hernia in children have drawn attention to the fact that usually the muscular development in the inguinal region is exceedingly good. I maintain that this is also true of young adults in whom hernia is of recent appearance. One finds that even in children who at birth are muscularly poorly developed, and in whom both umbilical and inguinal hernias are present, the muscular weakness rapidly disappears as the child grows older. The second source of weakness is the important one in adults, and will become more and more pronounced as the patient grows older. After a certain time the muscles in the inguinal region become so altered that return to the normal is impossible.

In longstanding cases in elderly men I admit that it may be necessary to perform that part of the radical cure usually known as the "second stage" of the operation, but in the majority of young adults it is not only unnecessary but harmful. No drawing together of the conjoint tendon and Poupart's ligament, however complicated the procedure, will ever be as efficient as Nature's method; and further, if this part of the operation is successfully carried out, it will prevent the normal valvular action of the internal oblique and transversalis muscles.

Those who have operated on recurrent cases of inguinal hernia, previously treated by "radical cure," will have noticed that the parts involved have lost all semblance to muscle structure, and have become a mass of fibrous tissue. If any one doubts the efficiency of the valvular action of the muscles at the internal ring let him, if he has not already done so, when operating on an inguinal hernia in a young adult, pass his finger through the ring, when the patient is straining should the opportunity occur. He will feel the vice-like manner in which the finger is gripped by the contracting muscles.

The present Professor of Systematic Surgery in Edinburgh has for long taught that the strength of the abdominal wall depends upon its mobility. Seaver (10) advocates exercises which strengthen the muscles guarding the internal ring as a method of treatment for inguinal hernia instead of operation. I believe in this method, provided that the neck of the sac be first dealt with. A patent funicular process will always be a source of danger, and it must be remembered that hernias frequently occur in young adults whose muscular development is excellent. It is quite possible that the sudden appearance of a hernia in a young adult during excessive exertion is due more to faulty muscular co-ordination than to actual muscular weakness.

I now pass to the third hypothesis: that if the neck of the sac be efficiently dealt with, it is quite unnecessary to interfere with the remaining portion in the inguinal canal or scrotum.

In dealing with that variety of hernia originally termed congenital, one has to divide the sac in the vicinity of the testicle. The level of this division varies with different surgeons.

In other varieties some surgeons purposely leave the lower portion of the sac *in situ*. Barker (11) divides the sac half an inch below the external ring, leaving the scrotal portion, the open end of which he ligatures. Maclaren (12) divides the sac at the same

level. Neave (13), in concluding a paper on hernia, says : " Sometimes it is better to leave the sac *in situ*, and only clear and divide the neck." Kocher (14) says if there is much matting the sac may simply be left alone as Wölfer recommends.

If a thickened and adherent sac can be left *in situ* surely there can be no harm in leaving the less abnormal ones. The first case in which I left the whole sac, having divided and ligatured the neck, was one on which I operated eighteen months ago. I have recently examined the patient and could not now tell that the sac had not been removed.

All the writers of the present day who advocate operation based on the saccular theory, emphasise the fact that the essential point of the operation is the complete removal of the sac. Clogg (6) says : " It is difficult to believe that they (herniæ depending upon the presence of a congenital sac) can be cured in any other way than by removal of this sac." I hold that obliteration of the neck of the sac is sufficient.

Why first attack the region of the external ring when it is really the internal ring we wish to get at ? The only reason I can see for this is that, in the past it has been considered essential to dissect out the sac, or at least the greater portion of it.

If we closely analyse the operations at present in use we are struck by the fact that they have only one thing in common—namely, obliteration of the neck of the sac at the internal ring. Further, we note that in recent years the tendency has been to make the skin incision at a higher level than has been customary in the past, but, as far as I know, there is no operation in which the internal ring is cut directly down upon without interfering with the parts below, although the aim of all is eventually to get at the internal ring.

In the past too much importance has been given to the technique of the numerous operations in vogue and too little to the principles involved.

Last month, on looking through some of the older literature on the subject, I found that Bennett (15) in 1893, having described the operation he recommends for inguinal hernia, mentions an operation somewhat similar to the one suggested this evening, but he limits its use to small bubonoceles, and even then only in exceptional circumstances. In slight cases of incomplete inguinal hernia he says it is " as a rule unwise under ordinary circumstances to make any attempt at effecting the radical cure at all." He

apparently takes it for granted that the sac in those cases is acquired. The incision is made over the most prominent part of the swelling; but if the hernia extends to the external ring he incises over that point in preference, strongly objecting to any division of the anterior wall of the canal.

Results of Operation

I have purposely entitled this paper a preliminary note as I have only performed the operation during the last eighteen months, and therefore sufficient time has not elapsed to allow of definite statements being made regarding the ultimate results, nor can I bring forward a large number of cases. The results up to the present, however, confirm my belief in the theories on which the operation is based, and are so satisfactory that I have thought it advisable to make this preliminary communication. I have performed the operation sixteen times—nine times in male adults whose ages varied from 19 to 40, the average age being 28; five times in male children; once in a woman aged 28; and once in a girl of 11. In three male adults local anæsthesia was used. I have either seen or obtained recent reports from all the patients; in every case the results are satisfactory. Without detailing the cases I will mention four of the earlier ones.

J. D., aged 24, developed a right inguinal hernia three years ago. He wore a truss until he was operated on a year and four months ago. He plays golf, dances etc., and has had no trouble since the operation.

A. S., aged 35, developed right inguinal hernia after running a race a little over a year ago. His work as a butcher involved lifting heavy carcasses, which did not improve matters. He was operated on ten months ago, got up in three weeks, and left hospital a week later. Since starting work, eight weeks after the operation, there have been no signs of recurrence.

J. W., aged 20, developed a left inguinal hernia after an attack of pneumonia eight years ago. He wore a truss at intervals until operated on ten months ago. He was up in three weeks and started his work as a grate-fitter four weeks later. The part operated on is now, to quote his own words, "as sound as a bell."

J. F., aged 30, suffered from an oblique inguinal hernia on the right side and had a patent tube on the left, although no hernia had ever been present. He was operated on eight and a half months ago. In six weeks he started light work as a "checker," and in another fortnight was playing three or four rounds of golf a week after working hours. Three and a half months after leaving

hospital he renewed his work as attendant at the Step Rock, St Andrews. He bathed daily, equalling previous records for swimming short distances; "trick diving such as somersaults, back dives, etc., presented no more difficulty than before." The following month he swam from the Step Rock to the pier, a distance of over a mile. At present he is perfectly sound.

Notes of the after-progress of the other cases are similar to the above.

Although this operation was primarily devised for young adults I have also used it in children. Dr Tuke of Dunfermline, to whom I spoke of it some time ago, has operated in this manner on eight cases, three adults and five children, all of which have done well. He has kindly allowed me to mention his results, and states his opinion that it is the ideal operation in children. It is difficult to state the age limit at which it would be inadvisable to employ this method. The decision rests on the possibility of the power of the muscles to regain their tone, rather than on the actual age of the patient.

In future, even in complicated cases, I intend to begin by exposing the internal ring and, if necessary, enlarge the incision downwards later.

The operation is easily and quickly performed. It can readily be done under a local anæsthetic, much more so than the operations usually employed, the nerves involved being under direct control.

Other advantages are that the patient need not be confined to bed for more than ten days or a fortnight. Once union of the overlapped external oblique has taken place, the sooner the internal oblique and transversalis muscles are brought into play the better; not only so, but the greater their development the less chance there is of a recurrence. In patients in whom the muscular development in the inguinal region is questionable, definite exercises should be started during convalescence. The incision is further away from possible contamination than that usually employed, this is specially important in children; but, on the other hand, the parts are smaller in children, and, therefore, at first the operation is more difficult.

Complications due to extensive dissections of the sac and manipulations or displacements of the cord are avoided, nor has hydrocele developed in any of the cases operated upon.

The overlapping of the external oblique, a method originally introduced by Lucas-Championnière (16), forms a firm pad over the internal ring without interfering with the action of the underlying

muscles. Fixation of the neck of the sac is unnecessary in children, but I have always done so in adults to prevent the possibility of any semblance of pouching at the internal ring.

If it be granted that oblique inguinal hernia in adults is primarily due to a preformed sac, then, from the clinical evidence we have in children, the operation which will deal with the sac with the least damage to the canal is what we must aim at. The operation I have suggested appears to me to carry out the above requirements in the simplest manner possible. Murray (7) says, "Clearness of purpose and simplicity of method should be our aim." I think you will admit the simplicity of method. I can only hope that I have succeeded in making my purpose clear.

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Meeting II.—December 4, 1907

Dr James Ritchie, *President, in the Chair*

I. Election of Members

William Fordyce, M.D., F.R.C.P., Ed., 20 Charlotte Square; J. Lamond Lackie, M.D., F.R.C.P., Ed., 1 Randolph Crescent; Peterswald Pattison, M.B., Parkhill, Colinton; and Arthur Murray Wood, M.D., D.P.H., 180 Ferry Road, Leith, were elected Ordinary Members of the Society.

II. Demonstration

Dr Aitchison Robertson gave a Demonstration of Milk treated by Dr Budde's process.

He said almost two years ago he had had the honour of submitting to this Society a communication on the Milk-Supply of this City, in which he indicated in what directions it might be improved.

He was much pleased with the interest which the paper evoked, not only amongst the Members of this Society, but throughout the country generally.

As regards any improvement in the conditions of our milk-supply, however, the suggestions might as well never have been made. The conditions of the cowsheds and dairies remain as unsatisfactory as ever. Complaints from consumers as to the dirtiness of the milk are constantly being made. Deposits of hair, mud, excrement may be obtained without difficulty from many samples, and examples much worse have lately come under my notice, for example, a clipping of a very dirty finger nail. A customer found recently a cream pitcher immersed in her supply of morning milk. A friend told me that he saw a little boy lift the lid of a can of milk standing at a door step and deliberately spit into it.

Such possibilities of contamination are scandalous and ought not to be possible.

As to tuberculosis in the cows of the Edinburgh cowsheds, Dr Aitchison Robertson was glad to know that it is apparently not so common as it is elsewhere. The Veterinary Inspector of Dairy Cows, Mr J. Riddoch, M.R.C.V.S., states in his Report that during the year 1906 he ordered forty-six cows to be removed from the byres; thirty-seven of these were found to be suffering from tuberculosis, and in nine the udder was invaded, and tubercle bacilli were present in the milk.

During the present year, forty-one cows were ordered out of the byres: twenty-seven of these were tuberculous, and in five the udder was the seat of the disease, and tubercle bacilli were present in the milk.

The tuberculin test has not been applied to any of the City cows. It would appear that our Municipal Authority will not take upon itself the control of our milk-supply, and if we were to have clean and safe milk we must look elsewhere for help.

Sterilisation of milk by boiling had the disadvantage that the constituents of the milk undergo a marked change, and though we do not believe that it produced scurvy-rickets (Barlow's disease) in infants and children by itself, yet the partial sterilisation of tainted by heating it to a temperature not exceeding 140° F., for twenty minutes, was to be preferred. The method was, however, troublesome, and would not clean the milk of extraneous matter.

Infants of tainted germ-free milk from their mother's breasts. Dr Robertson showed a Petri-dish containing gelatine agar, to which was added human milk obtained after merely washing the nipple with boiled water. It had remained sterile during the ten days it has been kept.

As a contrast here was another culture dish containing cows' milk as delivered in the morning. A count of it showed at least 565,000 organisms per cubic centimetre, which was far in excess of that allowable, viz. 30,000 per cubic centimetre. A sample of the same milk pasteurised for twenty minutes furnished no growths within three days, showing that most of the ordinary dairy organisms had been destroyed.

In his former paper Dr Robertson suggested that instead of making it a penal offence to add preservatives to milk, it would be advisable that the authorities should allow the addition of the minimal amounts of harmless antiseptics to milk, arguing that it would be better to run the slight risks of their injurious action if indeed they have any, rather than the undoubted risks which result from the use of such a putrescent fluid as ordinary dairy milk. Personally he did not share the belief that antiseptics such as boric acid or formaldehyde if used in the minimal amounts exerted any deleterious action on the animal economy.

Happily however another agent had been applied to the purification and preservation of milk. This was nascent oxygen as evolved from peroxide of hydrogen in contact with milk at a suitable and optimum temperature. Surely no objection on physio-

logical grounds could be taken against this agent, which is so easily decomposed into harmless components.

The addition of peroxide of hydrogen had been worked out most scientifically as well as practically by his friend Dr Budde of Copenhagen. He had now installed an apparatus to treat the milk on commercial lines, and milk so prepared might now be purchased at the ordinary market price of untreated milk. The culture plate which he showed was prepared from Buddeised milk, and had remained sterile during three days.

He had in numerous cases employed this milk practically in the rearing of infants as well as for general domestic use, and had in every case found that the milk was taken with relish and with benefit to the health of those consuming it.

The milk, besides being rendered germ-free, was cleaned and freed from blood, mucus, and extraneous matter. Dr Budde had, in his opinion, devised a plan which was eminently practical and one which fulfilled all the requirements of the strictest hygiene. That this was proved was shown by the fact that the Medical Officer of Health for Leith, Dr William Robertson, had introduced modified Buddeised milk for distribution to the poor instead of ordinary modified pasteurised milk.

He needed to say no more, but introduced Dr Budde as one who had done more for the future health of the infant population than any one whom he knew. He would describe the method which he had devised for the purification of cow's milk.

Dr Budde then said—

Mr President and Gentlemen,—I beg to thank you for your kind permission to put before you to-night some of my ideas about milk-supply. These I shall state as briefly as possible. Not being a medical man, I shall not waste your time by talking about things which every one of you is sure to know better than I. I just want to mention a few facts in order to make clear my arguments in favour of safeguarding the milk-supplies of our large centres of population.

Arthur Lux (*Centralblatt für Bakteriologie*, ii. 11, 1904) has carried out the most extensive investigations of bacilli in milk immediately after milking. He milked in sterile test-tubes, and the average of results of 270 tests was that the milk contained about 1200 germs per c.c. Such good results, however, are unattainable in ordinary practice. What can be attained in practice may be judged from a milk sold by a big company in Copenhagen, under

the name of "children's milk," and at a higher price than ordinary milk. It is supplied from cow-stocks under the strictest possible inspection, and where any suspicious cow is promptly removed. It is further milked directly down into milking-pails provided with ice-jackets and immediately distributed in sterilised bottles. According to V. Bie, this milk contains on an average about 70,000 germs per c.c., and it always contains such bacilli (*coli-bacilli*, *Staphylococci*, *Streptococci*) as may produce digestive derangements in infants.

In large British towns such fine milk is not available. The cows and the trade-milk are not uniformly inspected, much of the milk coming from country districts being unsatisfactory. The tuberculin test is sometimes used as a trade advertisement, but is often carried out in such a way as to be practically valueless. Many farms and small farmers are not particularly clean, and I know from personal experience that only very few farmers go so far as to filter the milk, although many of them are supposed to do so. The average dairyman is demoralised in so far that he does not realise his responsibility towards the public. His only care is to get his milk sold before it goes sour. What sometimes happens at railway-stations and on milk-cars in the streets is simply appalling. The result is that trade-milk always contains hundreds of thousands, often hundreds of millions of germs per c.c.

I need not mention to you the dangers of the tubercle virus so frequently found in trade-milk. This danger has been strongly emphasised by E. von Behring, who said in his lecture at Cassel on the 27th of September 1903: "Milk drunk by infants is the principal source of consumption," and the extensive investigations of the Royal Commission on Tuberculosis place it beyond doubt that this danger is very real. Of all diseases transmitted by milk, tuberculosis is undoubtedly by far the most frequent, and at the same time the one least realised by the public, because, in the majority of cases, it develops so slowly that the source escapes notice. This particular evil ought, of course, in the first place, to be attacked at the root — viz., the diseased cows and milk-handling persons — but this is not being done systematically as yet, and many years are bound to elapse before trade-milk is rendered safe to the consumer; and even with this original source of disease eliminated, there would still remain the danger of contamination of the milk during transport.

Infants reared in the natural way by healthy mothers get milk which contains very few germs. The same conditions should un-

doubtedly be provided for those infants who are so unfortunate as to find themselves reduced to artificial feeding. Also for adults the risks of taking germ-crowded milk are by no means to be overlooked.

As all of you know, the ordinary way of destroying germs— viz., by heat—cannot successfully be applied to milk, because a temperature which is sufficiently high to check the growth of the milk-germs, at the same time alters the character of the milk to such a degree that the product after such treatment is not entitled to the name of milk. The well-known results, as far as infants are concerned, are rickets and weakened economies without any power of resistance against disease.

I will, with your permission, shortly describe the scheme by which I propose to solve the difficulty in question.

The milk should be brought in from the various cow-stocks to a central place immediately after milking. Cooling is a dangerous thing if it be carried too far, because it destroys the natural anti-bacterial qualities of milk. Cooling to ordinary water-temperature is to be recommended if the milk cannot be dealt with at once.

Immediately after arrival the milk should be passed through a heater, where it is heated to 50° C. (122° F.), and from there through a centrifugal cleaning-machine of the same construction as a separator but with only one outlet, so as not to separate the cream from the milk. This machine not only removes all particles of dirt in a far more effective way than a filter, but it also removes many bacteria adhering to the particles of dirt and to the minute motes of the tissues of the cow which are always present in milk. The bacilli thus adhering to different particles have been experimentally proved to be the most resistant, which fact seems to be a mere question of mechanical protection. In order to explain this I might just remind you of a well-known experiment. Sew, for instance, bacillus anthracis on a piece of woollen thread, fasten the centre of it between a pair of tweezers, and hang the tweezers inside a steam-boiler. After some time you will find the ends of the thread sterile, whereas the bacilli from between the tweezers are in full vigour. It is very likely for a similar reason that, for instance, natural tuberculous milk, in which disintegrated udder-tissue is abundant, is far more difficult to sterilise than milk to which cultures of bacillus tuberculosis have been added. It is a well-known fact that the natural tuberculosis milk cannot even be satisfactorily sterilised by boiling. I, therefore, consider the centrifugal machine a very important part of any milk-treatment. After the milk has passed through the

machine the dirt, etc. from the milk may be seen adhering to the inside of the bowl.

After this the milk should be passed into a vat provided with water-jacket, the temperature of which can be raised and maintained by introduction of steam. As soon as, or immediately before, the milk has got into the vat it should be mixed with a little peroxide of hydrogen.

This, in small quantities, is a perfectly harmless substance. Every living cell contains an enzyme, isolated for the first time by Oscar Loew at Washington (1901), and by him named Catalase. Its characteristic quality is its power to decompose hydrogen peroxide into water and oxygen, and its presence everywhere excludes the possibility of hydrogen peroxide ever being found in a living cell (Loew, U.S.A., Department of Agriculture, Report No. 68, Washington). Consequently, hydrogen peroxide in contradistinction to all other antiseptics cannot be absorbed by the organism, but is invariably decomposed before it gets through the walls of the intestinal canal. The harmlessness of hydrogen peroxide taken by the mouth has also been established by numberless experiments.

The milk (often described as a "fluid tissue") also contains a certain amount of catalase. The proportion of this enzyme in milk may be estimated to something like 0.000001 per cent. When hydrogen peroxide is added to milk at about 50° C. (122° F.), it is able to decompose about 0.035 per cent. H_2O_2.

The hydrogen peroxide possesses in itself a strong germicidal power, according to Valdemar Bie, so strong that 1000 bacteria can be destroyed by 0.000000084 gram. H_2O_2. But the milk catalase causes a still stronger bactericidal action to take place, which arises from the formation of oxygen *in statu nascendi* (monoatomic oxygen), and which I have named the secondary germicidal action of hydrogen peroxide. I proved the existence of this secondary effect by comparing the action of hydrogen peroxide with that of any other disinfectant. I thereby found that all other antiseptics keep the milk fresh for a longer time at ordinary than at a slightly elevated temperature, whereas the reverse is the case with hydrogen peroxide.

I heat the milk to 50° C. (122° F.), because I found that this temperature was the optimum for the secondary effect, and is not high enough to alter the qualities of the milk. Professor E. von Behring, on my request, kindly investigated this question and established the correctness of my result.

The vat in which the milk is mixed with hydrogen peroxide

is preferably made of glazed earthenware, so that its inner surface is perfectly smooth. It is provided with a mechanical stirring-arrangement which is worked now and then in order to create and maintain an homogeneous mixture. The milk is kept in this vat at the same temperature for three hours, after which it is filled through sterilised tubes into sterilised bottles. The milk should be instantaneously cooled, which may be done either before or after the bottling.

I was able to prove that the amount of H_2O_2 the milk is able to decompose is also sufficient to bring about complete sterilisation. But the presence of catalase is not absolutely uniform. The safest course is, therefore, to add so much peroxide of hydrogen that there is certain to be a trace left at the end of the process. Then immediately before the bottling a few drops of a catalase-solution are added which destroy that trace.

I may mention by the way that H_2O_2 has the great advantage that (according to Sieber and Loewenstein) it destroys toxines but leaves antitoxines quite unaffected. In, for instance, a neutral mixture of diphtheria-toxine and antitoxine the latter may be set free by addition of hydrogen peroxide. Further, the presence of H_2O_2 in milk is very easily detected by the potassium-iodide-cum-starch test, by which a quantity as low as 0.0002 per cent. H_2O_2 is sure to be found.

The hydrogen peroxide used for this process must, of course, be chemically pure and is produced by distilling the so-called "medical" peroxide of the trade after the acids—always left in it in order to make it keep—have been carefully neutralised with caustic soda or lime. The catalase may be prepared from nearly any vegetable or animal substance by extraction with water, precipitating with sulphate of ammonia or alcohol, filtering, drying, and again extracting the residue with water. A more stable solution is obtained by dissolving the catalase in a mixture of glycerine and water instead of pure water. I have brought with me samples of both the catalase-extract and the distilled peroxide.

Milk after having been subjected to the foregoing treatment possesses the following qualities :—

None of the component parts of raw milk is in any way altered.

The milk is practically sterile and perfectly so in regard to the specific pathogene micro-organisms.

The milk is able to keep longer than ordinary milk, but notwithstanding this fact it should, of course, like all milk be consumed

as fresh as possible. This keeping quality of the milk is rather interesting, as it has been obtained without adding boric acid or other preservative remaining in the milk.

The milk is absolutely free from foreign substances.

As for the milk-enzymes, the tryptic and peptic proteases and the lipases are unimpaired. The oxydases are destroyed, but seeing that these do not occur in human milk, this is generally considered of no importance. The catalase is destroyed. On the other hand, oxydases nearly always accompany catalase, so that by the final addition of a little catalase both these classes of enzymes are restored to the milk.

The nutritive qualities are unaltered,and numerous cases have been recorded by medical men in which people (both infants and grown-up people) have been doing very badly on raw or cooked (Pasteurised and sterilised) milk, whereas milk thus treated has agreed very well with them.

The taste and flavour of the milk is practically unaltered. This you may try for yourselves if you will take the trouble to taste the samples of treated milk I have brought here to-night.

I am very sorry that the time does not allow me to give to-night an account of the work done by others in the same direction, nor the experiments of the large number of scientific men in different countries, whom I have succeeded in interesting in the matter, and to whom I am highly indebted for their valuable investigations.

The behaviour of the milk-enzymes during the process has been investigated by Dr Sjöquist, Stockholm; Dr Th. Madsen, Copenhagen; and Professor Julius Stoklasa, Prague. The summary result of their investigations and my own observations is mentioned above.

The bactericidal side of the method has been investigated by Dr Levin, Stockholm; Professor Fischer, Berlin, and his two assistants, Dr Emmerling and Dr Abderhalden; Professor Tanner Hewlett, King's College, London; Dr S. Rideal, London; Professor V. Stein, Copenhagen; Dr Mstislaw Lukin, Moscow (experiments carried out at the University of Zürich); Professor Winkler, Agricultural College, Vienna; Dr Ernst Baumann, Magdeburg; Dr Wm. Robertson, Leith; and many others. Further special experiments on natural tuberculous milk have been conducted by Professor Joseph Svensson, Royal Veterinary Institute, Stockholm; Dr P. H. Santesson, Stockholm; Dr Ernst Peterson, Agricultural Institute, Ultuna (Sweden), and Dr Bertrup Hansen,

Silkeborg (Denmark) (the latter on request of the Danish National Antituberculosis Society). All these scientific men declare unanimously that non-sporing organisms and the vegetative forms of sporagene bacilli are killed by the process. Regarding the spores, there is some difference of opinion, which can in most, if not in all cases, be traced back to some defect in the method used. I am confident that the method in its present shape kills even spores with certainty. Successful experiments have been carried out with bacillus coli communis, bacillus typhosus, bacillus diphtheriæ, bacillus tuberculosis, bacillus anthracis, bacillus subtilis, bacillus enteritides sporogenes, bacillus acidi lactici, bacillus dyssenteriæ, bacillus mycoides, aspergillus niger, staphylococcus pyogenes aureau, streptococcus pyogenes, spirillum choleræ asiaticæ, Penicillium glaucum, and several others.

Clinical experiments have been conducted by a large number of doctors, mostly Danish and Swedish. Any bad influence of the treated milk has never been observed, but it has often been found to be better and easier absorbed than ordinary milk. The treated milk seems to be of therapeutic value in the case of certain diseases, as, for instance, typhoid fever, achylia gastrica, etc.

Dr Baumann, Magdeburg, has proved that pepsin and hydrochloric acid act considerably quicker on this milk than on ordinary milk.

The superiority of such milk to heated milk as a food for infants has been proved by many Scandinavian doctors and recently by Dr Wm. Robertson, Leith.

The superiority of milk trated by my method to Pasteurised milk as a food for calves and pigs has been proved by Mr R. V. Toll, agricultural expert of the Swedish Government.

The method now described is in use to a large and increasing extent in Denmark, and I am glad to say that it has been taken up by a Scottish Company, which has agreed to provide a really hygienic milk-supply without raising the sale-price.

It is evident that the problem of supplying large towns with pure milk can only be solved by big companies. The expenses of getting the best local milk, of watching the cow-stocks and the farmers, of keeping people with sufficient knowledge to take care of the milk, of procuring and running the necessary machinery for treating milk, washing and sterilising bottles, etc., could never be defrayed by small dairymen. Experience from other countries as well as my personal experience from this country has fully convinced me of this fact.

The limited time at my disposal will not allow me to go deeper into the subject on this occasion, but I have collected results of experiments and facts connected with my method in a little pamphlet which I shall be pleased to send to anybody interested in the matter. I would also be pleased to answer any questions and arrange to carry out any tests suggested by your society.

III. Original Communications

1. THE ACTION OF CERTAIN SALTS OF FORMIC ACID ON THE CIRCULATORY AND MUSCULAR SYSTEMS

By Alexander Goodall, M.D., F.R.C.P., Ed., Lecturer on Physiology, School of Medicine of the Royal Colleges; Examiner in Physiology to the Royal College of Physicians, Edinburgh; and

Isabel Mitchell, B.Sc., Carnegie Demonstrator of Physiology, Medical College for Women, Edinburgh

(From the Laboratory of the Royal College of Physicians, Edinburgh)

I. Introductory

The formates at the present time are receiving much notice. Points of interest are associated with their chemistry, and in the realm of therapeutics no up-to-date tonic is complete without them. It has been claimed for them that they stimulate the heart, vessels, bladder, and alimentary canal, that they increase the appetite, abolish fatigue, and exert a tonic influence on muscles. One enthusiast suggests that for the latter reason formic acid should replace alcohol among the working-classes. It has been stated that the formates are powerful diuretics, that they conserve youth, delay senescence, and that they cure rheumatism, tremors, and pneumonia. When administered to rabbits they increase their vivacity, while the endowment of the ant with formic acid explains its industry.

The local application of formic acid is said to cure lupus and inoperable cancer, and it has been recommended as a preservative of food. The physiological action of substances alleged to be possessed of so many of the attributes of an elixir of life seemed to us to be worth investigating.

The literature on the subject is for the most part unsatisfactory. The work of Clement (1) and Garrigue (2) can only be described as that of wild enthusiasts. Huchard (3), however, to a certain extent confirms their results, though he does so in more restrained phraseology.

A much more careful piece of work is that of Fleig (4). In the first part of his paper he criticises his French confrères so unmercifully and so thoroughly that he leaves subsequent critics very little to say. Indeed, had Fleig's paper received the attention it deserves the formates would never have attained the position in fashionable therapeutics they now have. Fleig immersed exposed loops of intestine in animals in Locke's fluid and noted the movements. Formate of soda added in varying proportions never affected the contractions in amplitude, frequency, or rhythm. Experimenting with the isolated heart of rabbits he never obtained an excitor or reinforcing effect due to sodium formate. On the contrary, concentrated solutions caused toxic effects, lessened amplitude, diminished frequency and arhythmia.

In the case of a nearly exhausted heart, however, calcium formate acted as a stimulant, just as any calcium salt would. The contractions of the gastrocnemius of rabbits set up by induction shocks were not affected by sodium formate.

Nerve energy direct and reflex was not stimulated by sodium formate. Ergographic records, carefully carried out, were negative in Fleig's observations, and he ascribes the results of Clément and Huchard to the effects of training and suggestion.

Among the purely clinical records the only observations of importance are those of Croom (5) in 1906, and Ker and Croom (6), published last June. They have given formic acid to patients suffering from diphtheria, and conclude that the results they obtained are distinctly encouraging, particularly in relation to the occurrence of cardiac failure and paralysis. They, however, themselves admit that standards of comparison for its merits are difficult to obtain in such a disease. They cannot confirm the diuretic effect observed by Clément and Huchard, and in this particular standards of comparison are definite. While their general verdict on formic acid is favourable, it is expressed with an amount of Scotch caution which contrasts strongly with the enthusiasm of the French advocates.

The salts we employed in the following experiments were those of sodium, potassium, and calcium. In many instances, however, the action of the two latter was evidently affected, sometimes to a preponderating extent, by the metallic ion, so that the bulk of our later investigations were carried out with the sodium salt.

A few observations were made with formic acid, but its powerful

local nocuous effects in causing tissue necrosis and increasing coagulability of the blood obviously rendered it unsuitable for such investigations as we wished to carry out.

We may note in passing that the formic acid, even in dilute solution, has a local effect on muscle. The muscle becomes more translucent and acquires a curious glazed appearance.

II. Observations on Animals

1. *Effects on Blood Pressure.*—Observations were made on 14 rabbits, fully anæsthetised with ether. In most cases several salts were employed. Tracings were taken from the left carotid, and the solutions were injected into the left jugular vein. The vagus nerves were cut in the great majority of cases. The doses varied from 10 to 20 m. according to the weight of the rabbits.

Sodium Formate.—

Injection of a 1 500 solution had no effect.

Injection of 1 100 solution in 4 instances, and of 1 50 solution in 2 instances, had no effect.

Injection of 1 25 caused a very slight fall of pressure in 3 cases, but in one instance had no effect.

Injection of 1 10 caused a slight fall, with immediate recovery in 5 cases.

Saturated solution caused a fall of 64 mm. of mercury, followed by a gradual rise to normal.

Potassium Formate.—

Injection of 1/1000 caused a slight fall of pressure, with immediate recovery.

Injection of 1/100 caused a fall of pressure, with recovery in 3 cases, and was negative in 3 cases.

Injection of 1/25 in all of 3 cases caused a very definite fall, with recovery gradual and not always complete.

Injection of 1/10 in all of 4 cases caused a fall practically to zero, and was immediately fatal in 3 cases. The fourth animal recovered only after artificial respiration.

Calcium Formate.—

Injection of 1 500 had no effect.

Injection of 1 250 had no effect.

Injection of 1 100 caused a slight fall in one case, a slight rise

in another, had no effect in a third, and stopped the experiment by clotting in two instances.

Two injections of 1/50 had no effect.

Injection of 1/25 caused a slight fall, with recovery in 2 cases, and had no effect on a third.

Two injections of 1/10 (saturated) solution tended to cause a slight rise. In one case a slight fall was caused. In each case clotting occurred and prevented further observations.

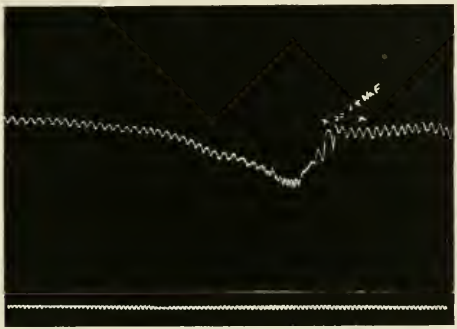

FIG. 1.—Blood pressure tracing from left carotid of rabbit, showing fall of pressure following the intravenous injection (between the two crosses) of 20 m. of sodium formate (1 in 5). Trace reads from right to left.

Formic Acid.—Two injections of 1/50 were made. In each case, after a preliminary fall and recovery, there was a long and gradual fall.

These experiments indicate that sodium formate has little effect on the blood pressure, unless it is in fair concentration. Strong solutions are definitely depressor.

Potassium formate is slightly depressor in dilute solutions, definitely depressor in moderate concentration, and usually fatally inhibits the heart in strong solutions.

The experiments with calcium were less definite, but they probably warrant the inference that the more dilute solutions have a slight depressor effect, and that concentrated solutions tend to raise the pressure.

The general effect of the acid in the two experiments was depressor.

The want of similarity in the action of the different salts is to be explained by the influence of the metallic ion. The true formate effect is shown in the case of the soda salt to be depressor. This depressor effect becomes enormous when added to the preponderating potash effect, while it tends to be obscured altogether when opposed by the stimulating action of calcium.

2. *Observations on the Blood-Vessels.*—These experiments were carried out on pithed frogs. A cannula was tied into the aorta and Ringer's solution was allowed to flow into it from a reservoir in which the fluid was kept at approximately the same level. The salt to be investigated was added to the Ringer's solution in known proportions. After flowing through the vessels the fluid escaped from a cut in the sinus venosus, and eventually dropped from the animal's toes. By such an arrangement any change in the calibre of the vessels affects the rate at which the drops fall. We used a graphic method for recording the drops. They were allowed to fall on a piece of tinfoil attached to the long lever of a receiving tambour. The movements were transmitted to the lever of a recording tambour and were marked on the smoked paper of a kymographion. Time intervals were recorded by means of an electric marker. After a salt had been perfused the vessels were again perfused with Ringer's solution only. Those experiments, in which the return rate most closely approached the original rate were obviously the most satisfactory, but a failure to obtain a return rate similar to the original does not necessarily invalidate the observation. Sixty-six experiments were carried out. The following is a typical example:—

Frog perfused with Ringer's Solution.

Original Rate . . .	20 per minute

After the addition of 1 in 1000 Sodium Formate.

In 30 seconds . .	22 per minute.
In 60 seconds . .	24 "
1 to 3½ minutes . .	28 "
3½ to 5½ minutes . .	26 "
5½ to 6 minutes . .	24 "
6 to 7 minutes . .	22 "
7 to 10 minutes . .	20 "
10 to 14 minutes . .	18 "

Ringer's Solution only.

–	14 to 19 minutes . . .	16 per minute.
–	19 to 20 minutes . . .	18 "

With a few exceptions it was found that all the salts in all the dilutions used first caused a dilatation, followed by constriction.

In 15 instances (7 potassium, 4 soda, 4 calcium) there was no dilatation, and in 10 instances (8 soda, 1 potassium, 1 calcium) no constriction occurred. In nearly every case the dilatation began within 3 seconds, never later than 30 seconds, and gradually approached its maximum. The constriction followed in from 17 to 480 seconds, usually about the expiry of a minute and a half. Although the intervals of time at which the dilatation and constriction occurred varied within very narrow limits, it seemed to us that in collating our results to select arbitrary intervals and state the conditions then found would not correctly indicate the result of our experiments. We therefore chose the maximum amount of change in each case.

The results are tabulated as follows.

The number of drops is that counted in 30 seconds.

Sodium Formate, 1 *in* 1000.

Experiment.	Original Rate.	Maximum Dilatation Rate.	Maximum Constriction Rate.	Return Rate.	Percentage Rate.	
					Dilatation.	Constriction.
1	108	127	99	99	117	91
2	20	22	14	19	110	70
3	19	19	14	19	100	73
4	33	47	16	..	142	52
5	136	151	114	130	111	83
6	12	16	10	10	133	83
7	142	..	115	171	100	81
8	38	56	56	56	145	100
9	34	37	12	12	109	35
			Average . .		118	74

Sodium Formate, 1 *in* 500.

Experiment.	Original Rate	Maximum Dilatation Rate	Maximum Constriction Rate	Return Rate.	Percentage Rate	
					Dilatation.	Constriction.
10	90	104	77	77	110	80
11	10	23	10	10	121	100
12	10	23	10	10	121	100
13	8	9	5	6	112	62
14	68	130	..	120	191	100
15	10	..	8	..	100	80
			Average	. .	126	87

Sodium Formate, 1 *in* 250.

16	77	96	71	71	124	78
17	10	26	10	10	136	100
18	19	25	16	16	131	84
19	7	10	7	7	142	100
			Average	. .	133	90

Sodium Formate, 1 *in* 100.

20	20	..	2	10	100	90
21	71	75	60	60	105	84
22	19	25	18	18	132	94
23	16	21	5	11	131	69
			Average	. .	117	84

Sodium Formate, 1 *in* 50.

24	60	180	60	60	300	100
25	18	24	10	18	133	55
26	11	19	9	9	181	81
			Average	. .	204	78

Sodium Formate, 1 *in* 25

27	60	166	60	60	276	100
28	18	29	4	..	161	78
29	9	11	4	..	122	50
			Average	. .	186	78

Sodium Formate, 1 *in* 10.

Experiment.	Original Rate.	Maximum Dilatation Rate.	Maximum Constriction Rate.	Return Rate.	Percentage Rate.	
					Dilatation.	Constriction.
30	5	7	..	6	140	100
31	19	39	12	15	200	63
			Average .		170	81

Potassium Formate, 1 *in* 1000.

32	188	..	125	136	100	66
33	10	11	7	10	110	70
34	171	..	129	156	100	75
35	56	..	48	56	100	85
36	45	47	39	42	104	86
37	60	67	36	55	112	86
38	33	46	17	26	139	51
39	9	12	10	9	133	100
			Average .		112	77

Potassium Formate, 1 *in* 500.

40	6	11	5	7	183	63
41	120	148	36	120	123	30
42	42	44	18	38	104	41
43	44	59	36	55	134	81
44	50	59	47	50	118	94
			Average .		132	62

Potassium Formate, 1 *in* 250.

45	8	8	3	8	100	37
46	30	34	7	26	113	20
47	55	..	25	40	100	45
48	50	57	39	51	114	78
			Average .		107	45

Potassium Formate, 1 *in* 100.

49	26	34	2	20	130	17
50	40	..	21	..	100	52
			Average .		115	34

Potassium Formate, 1 *in* 50.

Experiment.	Original Rate.	Maximum Dilatation Rate	Maximum Constriction Rate.	Return Rate.	Percentage Rate	
					Dilatation	Constriction.
51	20	..	3	..	100	15
		Potassium Formate, 1 *in* 10.				
52	25	20	13	10	115	52
		Calcium Formate, 1 *in* 1000.				
53	130	..	10	..	100	8
54	10	..	6	8	100	60
55	156	158	65	68	101	41
56	56	..	4	58	100	7
57	51	93	41	52	183	81
58	50	58	37	40	116	74
			Average . .		116	45
		Calcium Formate, 1 *in* 500.				
59	50	60	23	40	120	40
60	10	30	12	15	100	78
61	27	30	24	27	133	88
62	7	10	5	7	142	71
			Average . .		130	71
		Calcium Formate, 1 *in* 250.				
63	15	20	..	15	133	100
64	8	8	3	..	100	37
65	15	19	11	7	126	57
			Average . .		120	64
		Calcium Formate, 1 *in* 10.				
66	7	10	5	..	142	71

These tables show (1) that the sodium salt caused the greatest individual amount of dilatation, that the average amount of dilatation is greater than in the case of the other salts, and that sodium formate has the least constrictor effect.

(2) The potassium salt caused the greatest individual amount of constriction, the greatest average amount of constriction, and the least individual and least average amount of dilatation.

(3) The calcium salt had effects on the vessels intermediate between those of sodium and potassium.

(4) As regards strength of solution it is seen that in the case of soda and calcium the amount of dilatation increases in proportion to increasing strength of solution, and that the amount of constriction diminishes with increasing concentration.

In the case of potassium the amount of dilatation remains constant for all the concentrations used, while constriction increases with increased strength of solution.

3. *Observations on the Heart.*—(A) *Perfusion Experiments.*—Frogs' hearts were used. The perfusions were carried out by means of Schafer's heart plethysmograph. In some of the cases instead of using the piston recorder of Schafer's instrument we attached the very delicate and simple volume recorder recently introduced by Dixon (7).

Sodium Formate.—Seven observations were made with 1 in 1000 solution.

In two cases results were negative.

In three instances the heart was slowed and then stopped in diastole.

In two the heart became irregular and was stopped in diastole. In the latter five cases the force of contraction was greatly diminished.

Sodium formate 1 in 100 either perfused through the heart or merely painted on it, rapidly decreased the force of contraction, and in four out of five cases stopped the heart in diastole.

Sodium formate 1 in 10 caused great irregularity, with frequently an increased rate and speedy stoppage in diastole.

Potassium formate 1 in 1000, 1 in 500, and 1 in 100 caused irregularity, diminished force, and stopped the heart in systole.

Potassium formate 1 in 10 immediately stopped the heart.

Calcium formate 1 in 1000 augmented the force of the heart, sometimes caused irregularity, and soon stopped it in systole.

Solutions of 1 in 500, 1 in 100, and 1 in 10, slowed and stopped the heart in systole.

Formic acid 1 in 2000 diminished the force of contraction, and stopped it in systole.

Hearts perfused with formic acid showed a remarkable increase in translucency, in some cases becoming glassy-like and almost transparent.

Some points of interest emerged in the inter-relationship of the action of these salts. Thus a heart stopped in diastole by sodium formate could sometimes be made to beat again by perfusing calcium formate, but only for so long as the calcium formate was perfused.

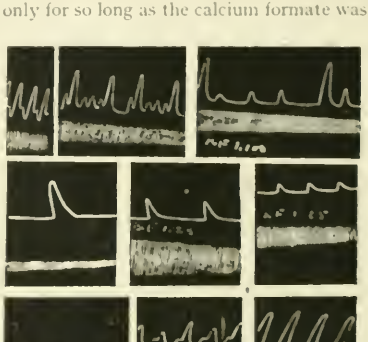

FIG. 2.—Frog's heart trace. 1. Shows effect of painting potassium formate (1 in 150) on the heart. 2. Shows effect of sodium formate on a heart nearly stopped by potassium formate. 3. Shows effect of painting calcium formate on the same heart.

A heart brought to a standstill by dilute calcium formate could be made to beat again for a brief period by perfusing strong sodium formate through it.

(B) *Heart Movements.*—These observations were also made on frogs' hearts. After the frog was pithed a hook was passed through the tip of the ventricle and attached by a pin to an aluminium lever writing against a smoked drum. The salts were applied to the heart by dropping the solutions from a glass pipette. Fifteen such experiments were carried out, the results were confirmatory of the foregoing. Thus, solutions of potassium and sodium formate caused arhythmia, followed in succession by diminished rate, diminished force with increased rate, diminished force with lessened

rate, and speedy stoppage with strong solutions. The potassium salt acted more powerfully than the sodium. Calcium formate 1 in 100 produced slight slowing of the heart only. Solutions 1 in 10 not only slowed the heart but lessened its force of contraction.

The application of calcium formate in several instances caused a return of contraction in hearts stopped by the application of the potassium or sodium salts. The two series of observations indicate that all three salts are toxic to the heart. In the case of potassium formate the toxic action is practically exclusive. In the case

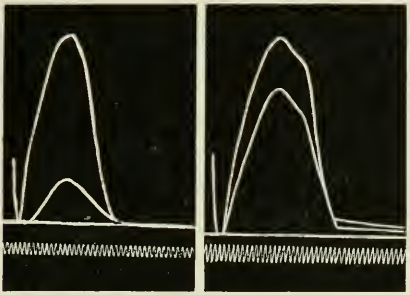

FIG. 3.—Left figure shows gastrocnemius contraction before and after injection of sodium formate (1 in 100) 3 m. into the muscle. Right figure shows the contraction of the opposite gastrocnemius before and after injection of 3 m. of Ringer's solution. The second curve is the smaller in each case.

of sodium formate a certain stimulating effect, probably in virtue of irritation, can also be noted. The effect of calcium formate is mainly that of calcium, the toxic effects being secondary.

4. *Observations on Muscle Curves and Fatigue.*—The gastrocnemius of the frog was used. The muscle of one limb was made to record the simple contraction induced by an induction shock of optimum strength, and was then either injected or painted with a formate. The corresponding muscle of the other limb was treated similarly with Ringer's solution. The same apparatus was used for the two limbs, the order of making the contrasted observations being alternated in successive experiments. In every case the muscle was loaded with 30 grammes.

It may be mentioned here that in no case did the Ringer's solution materially alter the height of the curve or the length of the latent period. Only the fatigue effects therefore need be stated.

Sodium formate 1 in 1000. Muscle injected with 5 m. Curves before and after were the same height. Completely fatigued after 600 shocks. Ringer preparation fatigued but still active.

1 in 100. Muscle injected with 5 m. Curve lowered 1 mm. Completely fatigued after 450 shocks. Ringer preparation showed only lengthening of latent period.

1 in 10, 5 m. Curve lowered 6 mm. Latent period greatly increased. Fatigued after 200 shocks.

Saturated, 5 m. Curve lowered 8 mm. Latent period increased. Fatigued after 150 shocks. Ringer muscle not affected

Potassium Formate.—

1 50. Curve lowered 2.5 mm.—fatigued after 400 shocks
Ringer preparation—fatigued after 800 shocks.

1 25 Curve lowered 3.5 mm.—fatigued after 250 shocks
Ringer muscle—fatigued after 600 shocks.

1 10. Curve lowered 5.5 mm.—fatigued after 100 shocks.
Ringer muscle—fatigued after 600 shocks

Saturated.—No response after injection.

Calcium Formate.—The height of the curves was somewhat diminished, but a great prolongation of contraction period became evident, due to calcium effect

Formic Acid—1 100 painted on the muscle lowered height of curve by 1 mm. Fatigue occurred after 200 shocks.

Ringer muscle—fatigued after 600 shocks.

These experiments indicate that the effects on skeletal muscle are similar to those on cardiac muscle—namely, that all three are toxic in the order, potassium sodium, calcium, but that in the case of the latter the toxic effect is subsidiary to the action of the metallic radicle.

III. Observations on the Human Subject

Although we now felt convinced that the administration of formates might be harmful, we made some observations upon ourselves, upon a lady student, and a laboratory boy. Conditions as regards work and exercise were practically constant during the experiments. Three of the subjects were habitual abstainers from alcohol and tobacco: the fourth acquired these virtues for a month previous to the experiments, and during their course. Only sodium

formate was used. The dosage was one gramme given thrice the first day, two grammes thrice on the two succeeding days, and once on the morning of the fourth day—a total of 17 grammes.

1. *Subjective Sensations.*—These were absent, except in the case of the student, who either misunderstood, or disregarded instructions and took the salt in an unduly concentrated form, and, in consequence, experienced an increased sense of thirst. No increase of appetite was noticed.

2. *Observations on Blood Pressure.*—These were made for a few

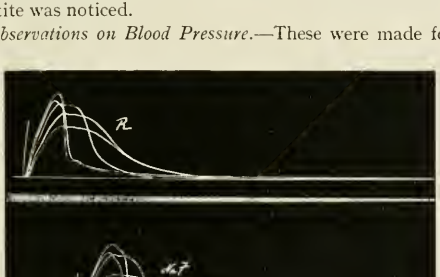

FIG. 4.—Fatigue curves. In the upper trace the muscle was bathed in Ringer's solution; in the lower, in Ringer's solution containing (1 in 1000) sodium formate. Each 150th curve was recorded. Note the more increased latent period and relaxation period in the formate preparation.

days before, during, and after the course of sodium formate, by means of the Riva-Rocci instrument, the reading being taken when the pulse became imperceptible at the wrist. The variations gave no indication of action on the part of the salt :—

	Day before formate taken.	Fourth day.
F. W. . . .	100	114
J. G. S. . . .	100	90
I. M.	110	98
A. G. . . .	110	114

3. *Ergographic Records.*—Mosso's instrument was used. A weight of 4.5 kilos. was raised over a pulley by flexion of the terminal

phalanx of the right middle finger every two seconds until complete fatigue occurred.

The time was marked by a metronome beating seconds. Three of the subjects of experiment naturally sustained the weight until the intermediate second struck, the fourth relaxed the muscle immediately after the pull. We thought it well not to attempt to alter this arrangement, and have made no attempt to place the records on an equal basis, as each only applies to the individual in question. It is particularly difficult to get satisfactory ergographic records for purposes of comparison. Training undoubtedly, and suggestion in all probability, play an important part. In order to eliminate the effect of training, two of the subjects practised with the ergograph daily until no further improvement occurred. The effects were remarkable. Unfortunately the early records in one case were not kept, but in the other there was an increase to eight times the original capacity for work in three weeks.

In the other two cases a single record was taken immediately before the formate course was begun, and a second record was taken four days later, when stiffness and pain on flexing the arm had disappeared. This method is probably not satisfactory, and it is interesting to note that after the formate course the untrained individuals showed improvement, the trained individuals showed diminution of work capacity.

Results :—

	Before	After 2nd day.	After 4th day.	After 5th day	After 6th day.	
F. W. (trained)	2.28	2.0	1.78	1.19	1.79	kilogrammetres
I. M. (trained)	12.08		10.19	..	..	,,
A. G. . .	4.02		4.41	..	..	,,
J. G. S. (contractions not sustained) .	10.23		21.02	..	..	..

Dynamometer Records. The observations were made with the right arm held at right angles to the body.

	Before Formate	After
F. W. . . .	30	20 kilograms.
J. G. S. . . .	34	55 ,,
I. M. . . .	30	30 ,,
A. G. . . .	57	55 ,,

These results all tend to show that in healthy human subjects sodium formate, in the doses given, has no appreciable effect.

Summary

Formic acid, formate of potassium, sodium and calcium in minimal active doses exert a toxic action on the circulatory and muscular systems. This action is enormously added to by the effect of the metallic ion in the case of the potash salt, and may be obscured by the metallic ion in the case of the calcium salt.

With the exception of calcium formate in strong solution, they all lower blood pressure. They all have a transient vaso-dilator, followed by a vaso-constrictor, action.

They are all toxic to the heart and to skeletal muscle in the order potassium, sodium, calcium, and accelerate rather than retard fatigue.

In the dosage stated, sodium formate has no demonstrable effect on the circulatory or muscular systems, or other noticeable effect on the healthy human subject.

Have the formates a place in therapeutics ?

In so far as our experiments afford an answer to this question (assuming that the formates are absorbed) we should say (1) that the medicinal use of potassium formate would be unjustifiable ; (2) that the effect of sodium formate might seriously impair the strength of a patient already debilitated ; (3) that in calcium formate we have a lime salt, freely soluble, and not too unpalatable, which has an action in coagulating the blood both in virtue of its metallic and basic ion.

We venture to suggest that in cases of remote or intractable hæmorrhage it is worthy of a trial.

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2. THE VALUE OF NOVOCAIN AS A LOCAL ANÆSTHETIC FOR SUBCUTANEOUS USE

By J. W. Struthers, F.R.C.S.Ed., Assistant Surgeon to Leith Hospital

There are now known a number of drugs more or less closely allied to one another, which have the power of paralysing nerve terminals or interrupting the conductivity of nerve trunks so completely that they may be successfully used to induce local anæsthesia for operative purposes. The most recently discovered of these is a synthetic product which has been termed novocain. Various advantages over similar drugs have been claimed for it, and it has in a short time attained great popularity, particularly in Germany, where it was discovered, or perhaps one should say elaborated, by Einhorn, and is now prepared in the laboratories of a well-known chemical firm. During the last few months I have made use of novocain clinically for inducing local anæsthesia by subcutaneous injection, and as I have found that the claims made for it seem well founded, I thought it might be of interest to members of the Society if I indicate briefly the evidence which my experience has afforded. My remarks are based on some eighty-six cases in which I have used novocain and carefully noted the results, contrasting them with those obtained from the use of cocaine and eucaine in some hundreds of similar cases. These results have been uniformly good, and although the number of cases may seem a small one to base an opinion on, I am inclined to believe that novocain is at least of equal and probably of greater value as a local anæsthetic than cocaine or eucaine for subcutaneous use.

In the first place, I may say that the drug is very soluble, and that its solutions are stable and may be repeatedly sterilised by boiling without in the least losing their power of inducing anæsthesia. I have tested this by making up a large quantity of a stock solution and using it over a period of several months, sterilising it over and over again during that time. The solutions combine well with solutions of adrenal preparations, and do not in the least interfere with the vaso-constrictor action of the latter.

For subcutaneous use, one requires, speaking generally, two solutions of a local anæsthetic—first, a relatively weak one for use in quantities up to several ounces, to produce anæsthesia by direct infiltration of the field of operation; second, a relatively strong one for injecting into or round nerve trunks such as the ulnar or

median nerves, to produce what is termed reginal anæsthesia in the area supplied by the nerve concerned.

For infiltration anæsthesia I have found that a solution of novocain in 0.75 per cent. saline solution of the strength of 1 : 400, plus 1 drop of the ordinary 1 : 1000 adrenalin solution to every 2 or 3 drms. of solution used, the strength of solution originally recommended by Braun,[1] answers admirably. It corresponds to what may be termed the standard solution for infiltration of 1 : 1000 cocaine, but has this advantage, that it may be used in larger quantities; for while the limit of safety is reached when about 4 oz. of the cocaine solution have been used, at least 6 oz. and probably more of the novocain solution may be used for an adult without any risk. In addition to this, it diffuses readily and acts as quickly as the cocaine solution, anæsthesia being satisfactory in ten to fifteen minutes after the injection is complete, and for this reason novocain is to be preferred to eucaine, for the latter may take as much as half an hour to take full effect. The duration of the anæsthesia is always more than an hour, often as long as three or four hours. After it has passed off, there is often, as with other drugs, a variable amount of burning and smarting pain in the wound, and I have seen no reason to infer that this is either greater or less than with cocaine, eucaine, etc. Sloughing of the skin, which occasionally follows the use of local anæsthetics, particularly, I believe, eucaine and stovain, I have never seen, nor have I seen it reported after the use of novocain.

I have used infiltration anæsthesia with novocain adrenalin solution of the strength indicated in the following operations :—

Tracheotomy, skin grafting, application of actual cautery, plastic operation on eyelid, exploration of sinus for foreign body, relief of paraphimosis. Removal of — testis, varicocele, tunica vaginalis for hydrocele, prepatellar bursa, olecranon bursa, carpal ganglion, fatty tumour, angioma, congenital mole, sebaceous cysts, varicose veins of leg, adenoma of breast, small subcutaneous fibro-sarcoma, tubercular ulcers.

The list is, I think, fairly representative of the class of operations which may suitably be done under local anæsthesia with success. A number of them have been done several times, and the total number has afforded a satisfactory test of the efficacy of novocain for infiltration anæsthesia. Several of them, it may be mentioned, were done in children as young as five and six years of age. In no case

[1] Braun, "Die Lokalanästhesie," Leipzig, second edition, 1907.

was there any sign of toxic symptoms arising from the use of the novocain adrenalin solution.

For regional anæsthesia a 2 per cent solution of novocain with 2 drops of 1 : 1000 adrenalin chloride solution to each drachm of solution used, is necessary when nerves as large as the median at the wrist or the ulnar at the elbow are being dealt with.

For anæsthetising digits by Oberst's method, the 2 per cent. solution may be used with perfect safety, but a 1 per cent. solution with adrenalin as before has been found quite strong enough to paralyse the relatively small digital nerves.

If a ring of this solution is injected round the base of a finger or toe into the subcutaneous tissue, the entire digit distal to the injection will be found anæsthetic in ten minutes. It was formerly the custom to apply a rubber band to the finger to localise the anæsthetising solution. The addition of adrenalin to solutions for inducing local anæsthesia, with the resulting anæmia and localisation of the anæsthetic action, has rendered the application of the rubber band unnecessary, and it is now never used.

The use of a local anæsthetic for anæsthetising digits by this method appears to me to afford perhaps as ready and accurate a method of comparing the relative strength of various drugs clinically as we possess, for the conditions in many cases are almost identical, and a given quantity of any drug can be accurately injected in each case and the effect easily watched and estimated. While a ½ per cent. cocaine adrenalin solution is strong enough to anæsthetise a digit completely in ten minutes, a 1 per cent. novocain adrenalin solution is required to ensure anæsthesia in the same time. While one would, however, hesitate to use more than 4 drms. of a ½ per cent. cocaine solution, as much as 6 drms. of the 1 per cent. novocain solution may be used without any risk. In point of fact such a quantity of a 1 per cent. solution is rarely required.

It will be noted that the doses indicated have not been stated as so many grains or centigrammes of novocain, but in drachms or ounces of the solutions recommended for use. This has been done in order to emphasise the fact that in stating the safe dose of any local anæsthetic, the strength of solution used must always be indicated, for a given quantity of novocain, cocaine, or other drug is much less toxic in a weak than in a strong solution. The actual amount of novocain suggested as the maximum dose in 1 per cent. solution is just over 3 grs., while the amount in the ½ per cent. solution is over 6 grs.

As regards the use of novocain for inducing regional anæsthesia, I have used it with success in opening whitlows of all degrees of severity, for the removal of ingrowing toe-nails, of subungual exostosis, for the treatment of hammer toe by excising the head and part of the shaft of the first phalanx, for amputation of fingers at and distal to the metacarpo-phalangeal joint, for removing needles and other foreign bodies embedded in the hand or fingers, for the cleansing and stitching of severe lacerated wounds, etc.

It has proved as satisfactory for regional as for infiltration anæsthesia, and, in conclusion, I may say, that I believe the advent of novocain marks a real though perhaps slight advance in the possibilities attending the use of local anæsthesia by subcutaneous injection. It is stable, readily sterilised, unirritating, and efficient as a local anæsthetic when combined with adrenalin, and can apparently be used in doses to meet all requirements without any fear of serious toxic symptoms arising.

3. CLINICAL REMARKS ON TUBERCULOUS CHLOROSIS

By Alex. James, M.D.,Ed., F.R.C.P.Ed., Consulting Physician to the Edinburgh Royal Infirmary

When we meet with a case which presents in a more or less marked degree all the ordinary appearances of chlorosis, and which yet on examination of the blood reveals the number of red corpuscles and the percentage of hæmoglobin to be practically normal, we are very likely to find in it a history of past or present tuberculous disease. This condition, often occurring in young women, is one which long ago Trousseau recognised and called false chlorosis or tuberculous anæmia ; and although in recent times, when blood examination has become more of a routine procedure, the co-existence in tuberculous disease of an anæmic appearance with a practically normal blood count is quite recognised, it seems to me that in cases apparently of chlorosis this possible association of tuberculous disease often passes for long unrecognised. Inasmuch as the recognition of such cases is important from the points of view alike of diagnosis, etiology, pathology, and treatment, I believe that the following are well worth recording.

Case I.

A. H., aged 16, a French polisher, was admitted to Ward 31, April 9, 1907, as a case of chlorosis. She complained of shortness

of breath, palpitation, and swelling of the ankles, and stated that she had been ill for three weeks.

History.

Her family history was not very good. Her father had died, aged 35, from Bright's disease; her mother is alive and well. Of a family of four, two brothers had died, causes unknown, one sister was alive and well. Her surroundings at home seemed to be satisfactory; at work, however, she had been in a room with many others, and the air, she said, got very close. As regards previous illness, she gave a history only of suppurating glands in the neck at the age of 6 or 7. They were quite cicatrised. Her present illness, she said, began about three weeks before admission. With some pain and discomfort in the feet she noticed swelling which the doctor told her was due to dropsy. The pain gradually ceased, but the swelling remained until her admission. She also noticed palpitation and breathlessness, and stated that she had been getting thinner.

State on Admission.

Height 4 feet 10 inches, weight 6 stone 3 pounds. Her development and muscularity were poor. Some œdema of both ankles was present, and her appearance was one of pallor and extreme anæmia. The temperature showed slight irregularity.

Circulatory System.—The pulse was usually about 90 or 100. She had complained of shortness of breath, faintness, and palpitation. As regards physical signs, little that was abnormal could be detected. The heart was not markedly enlarged, the sounds were all closed, and, in spite of her pallor, the *bruit de diable* was not well marked. The red blood corpuscles numbered 5,200,000, hæmoglobin 70 per cent., white corpuscles 7,187. A differential count gives polymorphs 57 per cent., lymphocytes 30 per cent., basophiles 3 per cent., eosinophiles 1 per cent., the opsonic index was 0·9.

Respiratory System.—The patient had no cough, but gave a history of coughs coming on from time to time. Some weeks earlier the cough was very troublesome, and she suffered from pain in the chest on coughing and breathing. On careful examination of the lungs, slight shrinking of the left apex with a slightly impaired percussion note was made out. On auscultation nothing was detected except slight harshening of the breath sounds. With the screen the X-rays

afforded corroboration of the physical signs, for the left apex lighted up much less distinctly on inspiration than did the right. The urine was pale in colour, showing no trace of urobilin ; and as regards the integumentary system, all that had to be noted was that there existed slight œdema of both ankles.

In this patient, then, we had presented all the appearances of chlorosis, and yet a practically normal blood count. The differential count of the white corpuscles showed, as was to be expected, a relative increase of lymphocytes, and the opsonic index, for the estimation of which I have to thank Dr Ian Stewart, was within normal limits. Dr Ian Stewart also tested in this patient the effect of tuberculin inoculation on the opsonic index, and found a rise on the third day, without any previous fall. All this may be regarded as indicating that there is present no active tuberculous mischief going on. But the evidence of the old tuberculous neck glands, and the condition of the left apex, undoubtedly point to the presence of tuberculous disease, whilst the occurrence from time to time of cough, expectoration, and chest pain, make this all the more manifest. The case is therefore one of tuberculous chlorosis.

Case II.

Constance H., aged 18, a domestic servant, was admitted to Ward 33 on December 18, 1905, complaining of breathlessness, palpitation, stomach pain, and swelling of the legs, and stating that she had been ailing from this for the last two years.

History.

Her family history was not very good ; her father died of Bright's disease, her mother in childbed. She had one sister alive and healthy, and one brother healthy : but of her other three brothers one had Bright's disease, another had some form of paralysis, and another had died from consumption. She had been in service for three years, and as regards food and home surroundings had been well placed.

Previous Illness.—When she was two years old she had disease of the bones of the left foot. According to her account the bones were scraped, and the condition healed. At the age of 14, after vaccination, she seems to have developed a scaly skin disease, for which she was treated in the skin wards of the Royal Infirmary for some weeks. She left the Infirmary improved, but the con-

dition was very tedious, and did not completely disappear for a year or two after she left.

Present Illness.—This seems to have begun about two years ago. She and her friends noticed that she was getting very pale, the lips especially. On climbing a stair she had breathlessness, palpitation, and pain over the heart; she had also headaches. At this time also her digestion troubled her. She had stomach pain and vomiting, and at times what she vomited resembled coffee grounds. In spite of dieting, and pills for the bloodlessness, she never felt strong.

State on Admission.

She appeared a well-nourished girl 5 feet 5 inches in height, and over 10 stone in weight. She was very anæmic, the face, lips, and gums showing marked pallor, and she had slight œdema of both ankles. The temperature was normal.

Circulatory System.—The pulse was about 70. She complained of breathlessness, palpitation, and pains over the heart on any exertion. She was giddy when she held her hands above her head. Examination of the heart revealed evidence of slight dilatation, but the sounds were closed in all the areas; there was a slight *bruit de diable*.

Hæmopoietic System.—The lymphatic glands and spleen were normal. Blood examination showed red blood corpuscles, 4,000,000; hæmoglobin 95 per cent.; white blood corpuscles 9000. The film shows a proportionate increase of lymphocytes.

Respiratory System.—She had had occasional coughs, but gave no history of hæmoptysis. On physical examination all that could be made out was a shrinking of the right apex margin with harshening of respiratory sounds there.

Alimentary System.—For some time past she had been complaining of atonic dyspepsia, pain after taking food, with occasional vomiting. She was habitually constipated. The abdominal organs were all healthy.

Urinary System.—All that need be noted here is that the urine was of rather low specific gravity and rather pale.

This, then, was a second case admitted as chlorosis, in which on account of the high red corpuscles and hæmoglobin estimation, we were led to look carefully for evidence of past or present tuberculous mischief. As will have been seen, this evidence was forthcoming.

As regards the course of these cases of tuberculous chlorosis, there is no doubt that though more tedious than ordinary chlorosis, they yet with treatment recover fairly well. But an interesting point is that with renewed feelings of health and vigour, and with great, often very great, improvement in their appearance, there is practically no improvement in the blood count. Indeed, I have had several cases which, on leaving the infirmary looking and feeling well, had an even smaller number of corpuscles and percentage of hæmoglobin than on their admission. Such was the case with the first patient, for on leaving the Infirmary her red blood corpuscles were 4,300,000, and hæmoglobin 70 per cent. This naturally leads to the question What is the real blood condition in those cases ? Here we enter in the realm of theory, but the idea which naturally comes into our minds is that there is really an oligæmia —that is, a diminution in the total amount of the blood. On this theory we can quite understand how, on recovery, we get associated with increased volume of blood improvement alike in the appearance and in the nutrition and vigour of the patient, and yet that the hæmocytometer and hæmoglobinometer show little or no change.

This theory is also interesting because it recalls the old view that in those likely to become phthisical a relatively poor growth and development of heart and vascular system may be recognised. To this view I have always felt inclined, and it has been especially interesting to me to find, as have other observers, that skiagraphic examination in phthisical patients has often revealed a distinctly small heart.

Trousseau not only drew attention to the existence of this tuberculous chlorosis, but emphasised the fact that great care should be exercised as regards its treatment, inasmuch as the administration in such cases of large doses of iron might cure the anæmia, but was not unlikely to be followed by hæmoptysis, and in time an acute and rapidly fatal phthisis.

This is perhaps an old-fashioned notion, and that grand old man, Trousseau, acknowledged that it was even in his own day. In his lecture on the subject he, however, stoutly affirms its truth, mentioning as conditions which also require equally careful handling *fistula in ano* and leucorrhœa in phthisical subjects. All this is, of course, a side issue ; and except to say that I am also old-fashioned enough to believe that what Trousseau has said is in the main true, and even to add two other conditions to his list, namely, enlarged neck glands and appendicitis in the phthisical, I shall do nothing more

at present than report a case which bears out Trousseau's statements as regards the effects of iron in tuberculous chlorosis. As will be seen, it is different from the other two in that the blood count was really below the normal, but in many important respects it corresponds to the type of tuberculous anæmias.

Case III.

J. F., aged 17, by occupation a silver-chaser, but lately a worker in the slaughter-houses, was admitted to Ward 31 on October 31 1905, as a case of anæmia, and complaining of shortness of breath on the slightest exertion, and headaches.

History.

His family history was not good. He was the youngest of a family of thirteen, of whom eight were alive. Of those who were dead, at least two died of consumption. His home surroundings and food conditions were good. As a silver-chaser he was exposed to bad air, but lately in the slaughter-houses he had always been in the open air, although exposed to weather and draughts.

Previous Illnesses.—About eleven years ago he had some inflammation at the base of the left lung. Six years ago he was treated in the Infirmary for anæmia. Three years ago he had cough, expectoration, and hæmoptysis, and two years ago he was a patient in the Victoria Hospital for Consumption, Craigleith for six months. Shortly after leaving there he noticed bloodlessness beginning again, and this had got much worse during the last month.

State on Admission.

He was about 5 feet in height, and his weight (7 st.) was fairly good. He was very pale and anæmic.

Circulatory System.—He complained of dyspnœa on the slightest exertion, also of palpitation, with occasional pains over the precordia. Examination of the heart revealed no marked enlargement, but a systolic murmur was heard over all the areas, especially over the pulmonary. The *bruit de diable* was marked.

Hæmopoietic System.—The spleen and lymphatic glands were normal. Blood examination reveals: red blood corpuscles, 3,500,000; hæmoglobin, 45 per cent.; white blood corpuscles, 5000. A differential count showed: polymorphs, 71 per cent.; small mononuclears, 23 per cent.; large ditto, 5.5 per cent.; eosinophiles,

0.5 per cent. The red corpuscles showed slight poikilocytosis, and they did not stain well.

Respiratory System.—He had very little cough or expectoration, and repeated examinations of the sputum showed no bacilli. Examination of the lungs revealed evidence of shrinking at the left apex posteriorly. The digestive system was practically normal, as also the urinary, the urine showing no appearance of urobilin. The opsonic index, as ascertained by Dr Ian Stewart, was 1.12, and after inoculation with TR a slight negative phase was noticed, not sufficient, however, in Dr Stewart's opinion, to warrant the diagnosis of tubercle, but yet more marked than would be expected in a healthy person.

After-History.

After some seven weeks' treatment in the ward, with rest, fresh air, good food, and arsenic, he showed great improvement. He acquired a good colour, and his nutrition and general vigour increased, so that he was able to take his share actively in the ordinary work of the ward. With all this, however, it was noteworthy that his blood count showed practically no change.

He was sent to the convalescent home, but there, after a few days, he developed a cough, and spat up a large quantity of blood. He was at once sent back to the ward, and there we found him presenting again all the appearances of great anæmia, and with some dulness and crepitations at the left apex. From this condition he rapidly recovered, only, however, as strength returned and pallor disappeared, to suffer again from hæmoptysis. He died a few weeks ago, apparently from anæmia and debility, and for the last eight or nine months of his life his condition was very much like that of hæmophilia—that is to say, as soon as he felt well and developed a good colour, he had hæmoptysis. Dr Leslie Lyall, who saw him during the last months of his life, informed me that iron always brought this condition about.

Lastly, as regards treatment in those cases of tuberculous anæmia. As already stated, Trousseau held that iron was to be avoided, and that arsenic and general treatment by hydrotherapeutics, salt-water baths, etc., were good. In the main I agree with this, for I look upon the conditin as one which occurs in individuals who, as the result of hereditary or acquired causes, have given indication that they possess that low resistance power to the tubercle bacillus which is best described by the old-fashioned phrase "the tendency to

consumption." In what way and to what extent this blood defect may be salutary as regards tuberculous lung disease, we can only conjecture. My own idea is that it may be by inducing an air hunger, and so stimulating lung function and nutrition. Anyhow, when we reflect that the quality of the blood is quite good, we can understand that treatment to improve the health generally rather than the blood specially is what is required.

4. UNUSUAL SEQUELA OF HERPES ZOSTER (? POSTERIOR POLIOMYELITIS)

By ALEXANDER BRUCE, M.D., F.R.C.P.Ed., Physician, Edinburgh Royal Infirmary

In view of the attention which has been recently drawn, more especially by Déjerine, Thomas, and Laminière, to the lesions of the spinal cord subsequent to herpes zoster, and as a contribution to the symptomatology of presumptive lesions of the posterior cornu, the following unusual case appears to be worthy of being recorded:—

On the 2nd of May of the present year, Miss C., stationer, aged 54, began to suffer from herpes zoster on the right side at the level of the 7th and 8th intercostal spaces. The attack was a very severe one, greatly prostrated the patient, and compelled her for three weeks to remain in bed. When she was allowed to get up she could walk quite well at first, but after a few days she observed a degree of weakness and a numb feeling in the right leg. This numbness was first felt below the knee, and then gradually passed up the right thigh and the right half of the trunk as far as the lower limit of the herpetic eruption. It then affected the corresponding part of the left side of the trunk, and extended downwards along the left lower extremity as far as the foot. Thereafter it began to diminish, and in a fortnight had almost entirely disappeared, only a slight tingling and numbness of the toes of the right foot remaining.

During this fortnight the right foot felt constantly cold, but when put to the ground gave a sensation as if there were hot pipes under the floor. The lower part of the back also felt very warm in bed, as if the patient were lying on hot bottles. She also noted that the lower part of the trunk and lower extremities had ceased to perspire.

Two or three days after the beginning of the numbness she drew

the attention of her physician, Dr Thin, to her condition. On his first examination, Dr Thin discovered a degree of tactile anæsthesia over the right gluteal region and the upper part of the thigh. On a second examination made on June 12th by Dr Thin and myself, it was found that there was analgesia over the whole of the right lower extremity and of the right half of the trunk below the level of the herpes. It was stated also by the patient that her sensibility to temperature was defective—that when she put her foot into very hot water it felt as if it were cold. A careful examination showed that this thermoanæsthesia was present on the right side over the lower part of the abdomen and the right lower extremity, *i.e.* over practically the same area as that which was analgesic. Tactile sensibility, except over the area on the buttock and upper part of thigh already referred to, was perfect on the right side. The patient could feel the ground under her foot perfectly well. She was perfectly conscious of the position of her limbs, and could imitate with the left limb accurately any position into which its fellow on the right might be placed. There seemed to be no material diminution of muscular power while the patient was in bed, but when the attempt to walk was made there was a degree of weakness of the right lower limb.

The deep reflexes were exaggerated on the right side, and the plantar reflexes were extensor on the right side and flexor on the left. There was no affection of the sphincters.

As the patient was regarded as suffering from an unusual form of organic affection of the spinal cord, and as she could not be adequately nursed in her own house, her removal to the Royal Infirmary was recommended. She was admitted to that Institution five days later, on the 17th of June, and the following notes with regard to her case were made, with the assistance of my Resident Physician, Dr Hugh Wilson :—

The *family history* appears to be unimportant. Father died at sixty-three of some unknown cause ; mother at seventy-three of cancer in the abdomen. One brother is alive and well ; a second brother died of consumption.

Patient suffered from measles, scarlet fever, and smallpox as a child, and from dry pleurisy two years ago ; otherwise she has enjoyed good health, has had a comfortable home, light work, and sufficient food. She is a particularly intelligent, sensible woman, quite free from any of the stigmata of hysteria, and, apart from a somewhat exhausted look, evidently the result of her recent

illness, she appears fairly healthy. There is no cyanosis or œdema anywhere.

The cicatrices of the herpes form a zone about 4 inches broad round the right side of the trunk from the middle line in front to the middle line posteriorly over the 7th and 8th intercostal spaces.

The circulatory system showed no abnormality, except an accentuation of the second sound of the heart in the pulmonary area. The pulse-rate was 80 per minute. The pulse was regular in time and force.

The respiratory system was practically normal, with the exception of a possible slight rise of pitch in percussion of the right apex.

The alimentary system was normal.

The urine was acid, showed a specific gravity of 1018, and contained no abnormal constituents.

Nervous System.—Sensory Functions.—On the examination of the sensory system it was now noted that the dissociation symptoms observed on the 12th of June were no longer limited to the right lower half of the trunk and right lower extremity but had affected the corresponding parts of the left side in an identical manner. Careful testing elicited no loss of tactile or muscular sense on either side below a line slightly above the level of the umbilicus, with the exception of a small area over the right gluteal region and right upper part of the thigh. Here light touches were with some difficulty recognised. Elsewhere sensibility to touch was acute, and its localisation accurate. The muscular sense of both lower extremities was in no way impaired. Below the level of the umbilicus, there was complete loss of sensibility to pain and to temperature, as indicated in the accompanying Figures 1 and 2. Between the level of the umbilicus and that of the lower border of the herpetic eruption the loss of pain and thermal sense was not quite absolute.

There was a complete absence of perspiration over the lower part of the trunk and the lower extremities. The patient said that when in her usual health she did not perspire readily, but her skin was now unusually dry all over this area.

The abdominal and epigastric reflexes were not elicited on either side. The plantar reflex was extensor in character, with separation of the smaller toes (*signe d'éventail* of Babinski) on both sides. There was a marked increase of the knee-jerks, Achilles-jerks, and adductor jerks on both sides. Direct muscular irritability was also considerably increased. There was no ankle clonus.

Apart from the feeling of pain on the right side, due to the herpes, there was no girdle pain or feeling of constriction round the body.

Motor Functions. — While the patient lay in bed the strength of the lower limbs seemed to be fairly equal on both sides. It seemed somewhat impaired as tested by the power of pushing and drawing up the limb and by the movements at the ankles. The patient could not stand or walk without support. With support she could walk a few steps, then became greatly exhausted, and had to be put back to bed. The muscles of the lower limbs felt somewhat flabby. There was no inco-ordination of the movements of the lower limbs. The circumference of each thigh was 13½ inches, that of each calf 10 inches.

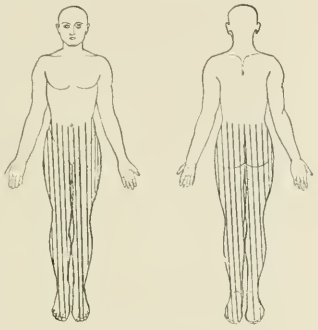


FIG. 1.—Vertical shading shows the area of analgesia on 12th June.

There was no loss of power in the hands or arms. The biceps, triceps, and supinator jerks were equal on both sides, and were not exaggerated.

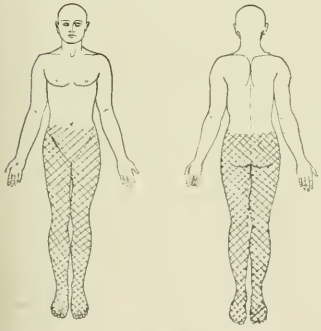


FIG. 2.—Area of anæsthesia to heat and cold on 12th June.

The functions of the cranial nerves were unaltered. There was no nystagmus. The pupils were equal and reacted to light and accommodation, and all the movements of the eye-ball were normal. The fundus oculi on both sides were normal. The field of vision was normal. There was slight pain on pressure above the left eye-brow.

The subsequent history of the patient during the time of her stay in hospital showed a somewhat rapid recovery from the

motor symptoms, and a more gradual and less perfect subsidence of the sensory phenomena.

On the 10th June it was noted that she began to perceive a pin-prick on the dorsum of the right foot as painful, and on the same area she could recognise a stimulus of very hot water as such, but could not recognise cold as such, or detect a slight elevation of temperature. Above the dorsum of the foot her sensory symptoms remained unaltered.

The general condition had considerably improved, but not sufficiently to permit of her being allowed out of bed.

In the evening, patient noted that for the first time since the beginning of her illness her feet began to perspire. The perspiration was copious, and made the feet feel wet and cold.

On the 20th of June the analgesia and thermoanæsthesia were lost as regards the dorsum of the foot, and slightly diminished between the ankle and the knee of the right leg. Here the prick of a pin, or a pinch, or the application of a test-tube containing very hot water caused a slight tingling sensation which could not be further differentiated.

On being allowed to get out of bed, patient could walk a few steps with support, but felt very insecure and became readily exhausted.

On the following day painful and thermal stimuli were readily recognised as such over the lower part of the right leg, and slightly over a corresponding area in the left leg.

On the 23rd of June an area extending forwards from the spinal column posteriorly for about four inches along the 9th, 10th, and 11th ribs on the left side was noted to be red and œdematous, slightly raised above the surface, and very tender to pressure. The colour over the area was fairly uniform, was not accompanied by vesiculation, and partly disappeared on pressure.

Pain.—A pin-prick was felt sharply below the knee on the right side, and faintly over the right thigh as high as Poupart's ligament. Over the lower abdomen, between the level of the umbilicus and Poupart's ligament, pain was not perceived on either side, and there was complete analgesia on the left lower extremity as far down as a level slightly below the knee. There was a narrow zone of hyperalgesia above the level of the umbilicus.

Temperature.—Below both knees, but especially on the right side, differences of temperature could be recognised fairly well. On the thighs, and on the lower part of the abdomen to a level

of about one inch higher than the upper limit of the analgesic area, heat was not recognised from cold. This level corresponded to a line drawn between the anterior extremities of the 9th ribs. Above this line a hot test-tube applied over the zone of the herpes caused both pain and a sense of heat.

Tactile Sensibility was quite normal, the area of partial anæsthesia on the right side having now disappeared.

Vaso-motor Symptoms.—A slightly heated test-tube caused ready and persistent flushing of the skin over the lower part of the abdomen on both sides.

Motor Functions.—Patient could now walk without support, taking short steps, evidently from dread of falling. There was no ataxia, and the muscular sense was quite normal.

During the following month the motor power in the lower limbs gradually improved, and does not call for special remark until about the beginning of August, when it was noted that the plantar reflexes had become definitely flexor in character. From this date up to the present time they have remained so. At no subsequent examination has any extensor response ever been elicited. The deep reflexes remained active, though not to the same degree as at first.

As regards the sensory functions, the tactile and muscular senses have remained throughout, and are at present (23rd November) absolutely normal.

The further history of the analgesic and thermoanæsthetic areas is as follows :—

The analgesic zone gradually contracted from below upwards. On the 3rd of July it was limited to an area from about one inch above the umbilicus to the level of the lower part of the abdomen. This area of analgesia remained more or less constant until she left hospital on the 5th of September. By the 23rd of November the area of complete analgesia had entirely disappeared.

During July and August the thigh and upper part of the leg showed somewhat varying degrees of sensibility to pain. Occasionally when the patient was tired during the examination the sensibility was found to be again lost, but the condition most frequently present was a diminution of the acuteness of recognising a pin-prick as painful. That condition in indicated in Fig. 3 (dotted vertical lines.)

At present (23rd November) the analgesia has entirely gone, but

the prick of a pin is not so acutely felt over the thighs and the lower part of the abdomen as elsewhere. As the analgesia diminished it was noted that stroking with a brush caused intense hyperæsthesia. This phenomenon gradually disappeared.

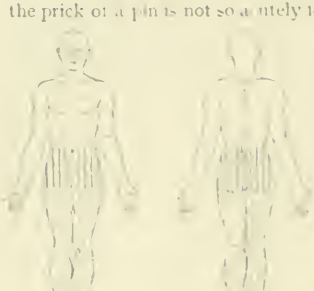


FIG. 3.—Vertical lines show area of analgesia found on 1st August. Vertical dotted lines show area to which it had receded by 9th August.

Temperature.—The perception of differences of temperature was somewhat irregularly regained. The area of thermo-anæsthesia contracted from below upwards. Cold was recognised as such before heat. By the 9th of August the zone of complete thermo-anæsthesia was limited to an area extending from the 9th rib to the level of the crest of the ilium. Below this a very hot test-tube caused pain and pricking as well as a sensation of heat, which was only recognised after some delay (Fig. 4).

On the 23rd of August it was noted that application of a cold test-tube applied to any part of the area which was primarily thermoanæsthetic caused a painful pricking sensation. On several occasions it was noted that there was an exaggerated sensibility to heat, cold, and to pin-pricks above the level of the herpes, but these exaggerations were very fugitive, and have now absolutely disappeared.

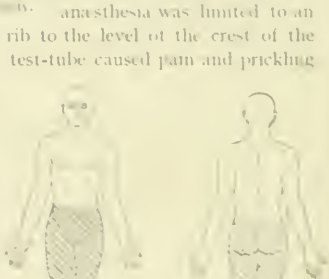


FIG. 4.—Shows area in which cold was recognised as heat. Outside this area it was correctly recognised.

Status præsens.—Patient has entirely recovered her power of walking. The epigastric and abdominal reflexes have returned. The plantar reflexes are both

flexor. The knee-jerks are somewhat active; the Achilles-jerk is present and active. There is no ankle clonus. There is no area of complete analgesia, although there is some diminution of the acuteness of pain-perception over the thighs and the lower part of the abdomen. There is slight confusion between the hot and cold test-tubes, but after a slight delay they can generally be accurately perceived except on the back of the left foot, where the cold tube is called hot and produces a painful stimulus. A very warm test-tube is well recognised as such, and also produces a painful cutting sensation.

Patient suffers greatly from troublesome paræsthesiæ; the thighs constantly feel as if burning, and the feet and lower parts of the legs as if encased in ice.

The general health is now very good.

Commentary

When the patient was examined on the 12th of June by Dr Thin and myself the following symptoms were present and had to be accounted for:—

(1) Muscular weakness of the right lower limb;
(2) Exaggeration of the deep reflexes;
(3) Extensor plantar reflex;
(4) Loss of pain sense in the right lower limb and lower part of the right side of the trunk;
(5) Loss of thermal sense in the same region;
(6) Conservation of muscular sense; and
(7) Conservation of tactile sense in the same region (with the exception of a slight area of anæsthesia over the buttock and thigh).

The motor symptoms were of such a nature as to postulate the existence of an organic lesion as distinct from a mere functional disturbance, and to point to a lesion, moreover, involving some part of the upper motor neurones.

The sensory phenomena were practically identical with the dissociation symptoms so often found in syringomyelia, and pointed to a lesion of the base of the posterior horn.

The fact that the motor and sensory phenomena were on the same side of the cord and not on opposite sides, as in the Brown-Séquard syndrome, indicated that the path for the conduction of

thermal and painful stimuli was involved before it had crossed to the opposite side of the cord, and further that the path from the opposite side was not involved.

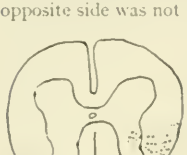


Fig. 5.—Degenerated fibres of the pyramidal tract extending into the base of the posterior cornua. (Sutherland Simpson and Jolly.)

The lesions in syringomyelia indicate that the loss of the conduction of painful and thermal stimuli with the conservation of the tactile and muscular sense depends upon a lesion at the base or in the substance of the posterior horn. A lesion, therefore, occurring in that situation would produce the sensory symptoms above noted.

The difficulty now arose of finding a point at which the pyramidal tract could be simultaneously involved. It is usually supposed that the pyramidal tract terminates by sending its fibres directly forwards and inwards towards the cells in the anterior cornua, but recent researches, in particular by Dr Sutherland Simpson and Dr Jolly (11) in Professor Schäfer's laboratory in the University of Edinburgh, demonstrate that, in the monkey at any rate, this is not the case, but that in the cervical, thoracic, and lumbar regions the fibres pass from the pyramidal tract directly inwards to the outer half of the base of the posterior cornua. The accompanying illustration (Fig. 51), published with the consent of the authors of the paper and the Council of the Royal Society, Edinburgh, demonstrates this fact very clearly. It will be obvious that a lesion involving the base of the posterior cornua in the area containing the degenerated fibres would produce both the sensory and the motor symptoms above mentioned. It will be noted also that such a lesion would almost certainly involve part at least of the intermedio-lateral tract, and be responsible for the interference with the sweat secretion present at the beginning of the illness.

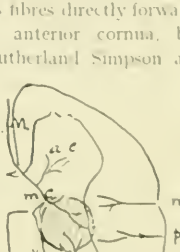


Fig. 6.—Diagram to show the [illegible] supply of the [illegible] of the posterior horn. Sutherland Simpson and Jolly [illegible] anterior and lateral [illegible] where [illegible] might affect [illegible] the pyramidal tract and [illegible] given and [illegible] cornua.

The lesion, from its gradual onset and limitation, was probably

of vascular origin, either thrombotic or inflammatory, arising in connection either with the postero-lateral or the posterior cornual arteries, or perhaps the mid-cornual branch of the central artery. Such a lesion causing a pressure upon and disturbance of function of the terminal fibres of the pyramidal tract, the cells involved in thermal and painful conduction, and those of the intermedio-lateral tract associated with the function of sweating and of vaso-motor control, would explain the symptoms completely (Fig. 6).

The subsequent extension to the other side could only be attributed to the occurrence of a similar lesion on the opposite side of the cord.

FIG. 7.—(Borrowed from André-Thomas and Laminière.) Shows hæmorrhage at the base of the posterior horn extending into the lateral horn.

So much attention has been paid to the symptomatology of the vascular lesions of the anterior cornua that the explanation of affections of the posterior and lateral cornua have been largely overlooked. In a communication by Dr Harvey Pirie and myself (2) in the *Review of Neurology and Psychiatry* at the beginning of this year, attention was drawn to the association of the intermedio-lateral tract in the dorso-lumbar region with the function of sweating in the lower extremities. Since the patient was first seen and the diagnosis made, an interesting paper has been published by André-Thomas and Laminière (3) in which vascular congestion, hæmorrhagic foci, and disturbance of the grey matter on the side of a herpetic eruption and in the region of the lateral horn are described and figured. I have taken the liberty of reproducing one of their figures (Fig. 7), which shows the occurrence and site of such a hæmorrhage, and it will at once be seen that were such a hæmorrhage to extend downwards into the lower dorsal and lumbar region it would account exactly for the symptoms of the disease.

The varying degrees of recovery of each function would depend upon the greater or less destructive action on the part of the original lesion.

Whether or not the view taken as regards the nature of the lesion be correct, the case is of interest as showing that herpes zoster is a condition which may leave effects more extensive than those due to the spinal ganglion in which the lesion originally occurred; and it will probably be found, on further investigation, that pathological changes are confined not merely to the posterior root-ganglion and to degenerations in the peripheral and sensory nerves consecutive to its destruction, as has been so well shown by Déjerine and André-Thomas [4], but that there are not infrequently wide-spread lesions within the cord itself.

LITERATURE

1. Sutherland Simpson and W. A. Jolly. "Degenerations following Experimental Lesions in the Motor Cortex of the Monkey." *Trans. Roy. Soc. of Edin.*, vol. xxxvii, No. 3, 1907, p. 2[illegible].

2. Alexander Bruce and J. H. Harvey Pirie. "A Plea for the Study of the Intermediolateral Cell-system of the Spinal Cord." *Ann. Neurol. and Psychiat.*, Jan. 1907, p. 1.

3. André-Thomas et Laignel-Lavastine. "Les Lésions Médullaires de Zona." *Rev. Neurol.*, July 30, 1907, p. 6[illegible]5.

4. Déjerine et André-Thomas. "Les Lésions radiculo-ganglionnaires du Zona." *Rev. Neurol.*, May 30, 1907, p. 627.

Meeting III.—December 18, 1907

DR JAMES RITCHIE, *President, in the Chair*.

I. EXHIBITION OF PATIENTS

This was a Clinical Meeting held in the Royal Infirmary.

1. *Dr Alexander Bruce* exhibited the following cases:—

(*a*) Syringomyelia of the spasmodic type, involving the left upper extremity.

(*b*) A hematrophy of the left half of the tongue, associated with lateral and rotatory nystagmus more marked on looking to the left side.

(*c*) A case of myasthenia gravis.

(*d*) An amyotrophic lateral sclerosis beginning somewhat acutely with hemiplegic symptoms and progressing rapidly.

(*e*) An aneurism of the left internal carotid artery with rupture into the cavernous sinus.

(*f*) A unilateral exophthalmos involving the right eye, associated with tremors in the right arm and leg, slight swelling of the right

side of the face and neck, free secretion of tears and sweat on the corresponding side, and great emotional disturbance, subsequent to double oöphorectomy.

(*g*) Case of neuritis of the left median nerve with tactile anæsthesia and hyperalgesia over the distribution of the median nerve, tenderness to pressure along all the nerves of the arm, the brachial plexus and the cervical plexus, as well as the second and third dorsal nerves; a considerable degree of atrophy of the muscles of the arm and shoulder, with exaggeration of deep reflexes, but without altered reaction to electrical stimulation.

2. *Professor Chiene, C.B.*, described—

(*a*) A patient, to show the result of an operation for lengthening the sterno-mastoid muscle in a case of congenital wry-neck. He did not bring the patient before them because the child was suffering from a very acute inflammation of the middle ear on the opposite side from the shortened muscle. The affected muscle was two inches shorter than the muscle on the sound side. He reflected a flap of skin from the squamous portion of the temporal bone; he then stripped off a rounded flap of periosteum from the mastoid process. The apex of the flap was two inches from the apex of the mastoid, the muscular attachment was also separated, and the apex of the periosteal flap was stitched to the apex of the mastoid process. The skin flap was then replaced. The muscle was thus lengthened, and the scar, being under the hair, will be invisible. The child can now move his head in every direction.

(*b*) He exhibited a case of loose body in the elbow joint, causing locking, and preventing the patient following his work as a joiner. A flap of skin was turned down from the lower third of the upper arm, the triceps was split, and free access obtained to the joint. The loose body was removed. A couple of catgut stitches brought together the edges of the split triceps, and the skin flap replaced. The movements of the joint are now perfect. It was suggested that a similar operation might be adopted in excision of the elbow-joint. It is interesting to note that there has not been, since May 1905, a case of disease of the elbow-joint requiring excision. Why is tubercular disease of this joint so rare nowadays?

3. *Mr F. M. Caird* exhibited—

(1) A series of patients who had been subjected to PYLORO-GAS-

TRECTOMY FOR COLUMNAR AND ADENO-CARCINOMA. The cases presented the usual clinical features in varying degree. Free hydrochloric acid failed in the test meals. According to the amount of the gastric organs removed it was possible to effect a direct implantation of the duodenum into the gastric sac (Kocher's method), or, closing both the divided stomach and the duodenum, to effect intestinal union by a gastro-jejunostomy (Billroth's second method). The patients are at present all in good health.

(*a*) William M., aged 48. Well marked family history of carcinoma. Operation, 16th Nov. 1907. Billroth's method.

(*b*) John B., aged 53. 16th Nov. 1907. Billroth.

(*c*) Mrs R., aged 66. 1st Nov. 1907. Kocher's method.

(*d*) Mrs C., aged 50. 31st Oct. 1906. Billroth.

(*e*) Mrs C. K., aged 43. 20th Sept. 1906. Billroth.

(*f*) Robt. E., aged 56. 16th March 1901. Billroth.

(*g*) Donald M'L., aged 32. 28th Aug. 1806. End to end.

(*h*) John M'L., aged 62. Well marked family history of carcinoma. 4th Aug. 1806. Kocher.

Drawings and preparations of the parts removed were also shown.

(2) Operation for IDIOPATHIC DILATATION OF THE COLON WITH OBSTRUCTION.

Mrs H. W., aged 38, was admitted suffering from CHRONIC OBSTRUCTION BECOMING ACUTE. The abdomen was markedly distended with dilated coils of intestine, and the diagnosis of a probable annular sigmoid stricture was made. Operation, 12th July 1907. A large hypertrophied dilated colon was found, rather kinked, in the pouch of Douglas. There was no stricture. An inguinal colostomy was formed to relieve the obstruction and fix the colon. The artificial anus was then dealt with on Sept. 14th, by forming an intraperitoneal lateral anastomosis, and as this still left a fæcal fistula which refused to close, the outlying knuckle of bowel was removed under cocaine, Dec. 3rd. The result is cure of the condition.

(3) Operation for ACUTE PANCREATITIS.

Mrs G., aged 27, who had for five or six months SYMPTOMS OF GASTRIC TROUBLE, was admitted five days after a severe attack of sudden epigastric pain. For four days there had been vomiting, and for three days no passage of flatus. She had rapid, catchy, painful respirations, but the general symptoms were not so distinct and pronounced as usually found in cases of perforated gastric

ulcer. Pulse 144; temperature 99; leucocytosis, 30,000. A diagnosis of possible acute pancreatitis was made. Operation, 21st Nov. 1907. Mesial laparotomy revealed a peritoneal cavity filled with blood-stained fluid, and the omentum showed well-marked fat necrosis, chiefly on its under aspect. The lesser omentum was lacerated, and a swollen, tense pancreas was seen, with two large, greyish-green areas. The pancreas was incised and drained with iodoform gauze and tube. The bile-ducts were normal. The gall-bladder was pulled to the mid-line and sutured to the peritoneum. On opening it dark tarry bile escaped, and numerous small, irregular, rounded gallstones. An incision was made above the pubes, two Keith's tubes were carried into the pouch of Douglas, and the peritoneal cavity well washed out with saline solution. The abdomen was then closed. The patient made a good recovery. Dr Chalmers Watson found Cammidge's test well marked on examining the urine.

4. *Dr R. W. Philip* exhibited—

A series of cases, illustrative of the value of tuberculin as a diagnostic. He recalled the fact that tuberculin had for many years been used subcutaneously from this point of view. He had used it regularly since its first introduction, and had found it of considerable value in doubtful cases, and it had in no single instance led to accident. Still it remained open to a certain objection in this sense, that, by the introduction of tuberculin into the circulation, a more general reaction was initiated than was actually necessary. The recently introduced cutaneous and ophthalmic methods obviated this objection. A definite and visible local reaction followed without any apparent general disturbance. The tests were especially serviceable in the case of young children, where frequently symptoms and physical signs were obscure. The tests were also serviceable as corroborative proofs where the tubercle bacillus was not determinable in any discharge, or where no discharge was available.

The cutaneous method, proposed by Von Pirquet, consisted in the application to a finely scratched area of skin of a drop of a solution of tuberculin (Old Tuberculin (5 p.c.), Acid Carbol., $\frac{\cdot\cdot}{aa}$ 25 parts. Normal Saline Solution, 50 parts). Within twenty-four hours, a small red projection appeared at the site of inoculation. During the following day, some infiltration was apparent, and a more or less definite papule resulted. The colour was at first

vividly red, tending to fade in the course of a few days. The infiltration disappeared; the patch might desquamate slightly. Thereafter some pigmentation remained for weeks. Occasionally the exudation was more marked, the skin being raised in wheal-like fashion. Sometimes a more vesicular form occurred. The reaction was occasionally delayed. Six cases were presented, showing the typical reaction at different stages, three or four inoculations having been made in the same individual at intervals. The cases included three of pulmonary tuberculosis in which T.B. could not be discovered in the discharge, a case of obscure abdominal tumour, which had been supposed tuberculous, a case of peritonitis, and a case of mitral stenosis and regurgitation with adherent pericardium and pulsating liver, in which a history of pleurisy some years ago and one or two slight physical signs raised suspicion of tuberculosis.

The ophthalmic method, proposed by Calmette, consisted in the application to the conjunctiva of a drop of dilute tuberculin. Five milligrammes dried tuberculin, precipitated by alcohol, in ten drops of sterilised water, or normal saline solution. In a tuberculous subject, a local reaction occurred in from three to six hours after instillation. The eye became red. The conjunctiva, especially the palpebral conjunctiva, was congested, and there was frequently some trace of œdema. The lachrymal caruncle became swollen. Sometimes a certain amount of fibrinous exudation appeared. There was little or no subjective disturbance, and the reaction gradually disappeared in the course of a week. Occasionally the reaction was delayed until eighteen to forty-eight hours after inoculation. Four cases were shown with typical reaction, including one of tuberculous peritonitis, one of cardiac disease, one of chorea, where, apart from the delicate aspect of the child, there was no apparent history or symptoms pointing to tuberculosis, save a few slightly enlarged glands in the supra-clavicular triangle, and one case of pulmonary tuberculosis in a young child, in which symptoms and physical signs were relatively slight and although expectoration was present no T.B. was determinable. In contrast with these cases showing positive reaction, two delicate looking children were shown, (1) a rachitic boy, with extra large abdomen, suggesting the possibility of tuberculous peritonitis, and (2) a young boy, subject to recurrent convulsions of the right arm, in whom the possibility of cerebral tuberculosis had suggested itself. In neither case had repeated use of the tuberculin solution produced any reaction.

Dr Philip quoted his own and other statistics to show that both tests were serviceable. A positive reaction occurred in about 95 per cent. of all cases, which, on other grounds, seemed likely to be tuberculous. In 75 to 80 per cent. of cases in which tuberculosis might be suspected, but in which no definite evidence was present, the reaction was positive, this group including a number of interesting cases, such as those of chorea and pericarditis, shown this afternoon. Curiously enough, both reactions sometimes failed to appear in undoubted cases of far-advanced tuberculosis, *e.g.*, pulmonary, meningeal, etc. This was in remarkable correspondence with what had been observed previously after subcutaneous injection of tuberculin.

5. *Mr Alexis Thomson* exhibited the following cases :—

(*a*) A patient, aged 53, operated upon for ACUTE INTUSSUSCEPTION CONFINED TO THE SMALL INTESTINE, and which being irreducible, the entire segment of gut involved, including the sheath, had to be resected. The preparation was shown at the meeting.

(*b*) A patient, aged 29, who had sustained a gunshot injury of the upper arm six months before he came under observation, with smashing of the humerus and tearing of the musculo-spiral nerve. An operation was performed for un-united fracture of the humerus, when it was found that the extent of destruction of the nerve rendered suture impossible. The distal end was therefore inserted into the median nerve below the elbow.

When shown to the Society six months after this operation, although he was able to use the arm at his work as a joiner, there still existed an amount of drop-wrist, for which Mr Thomson proposed to perform another operation, anastomosing the upper end of the musculo-spiral nerve with the median at a higher level.

(*c*) The preparation from a patient, aged 69, after a radical operation for CARCINOMA OF THE CÆCUM performed in two stages. The patient was so very feeble that an operation in one stage was not deemed justifiable. The result was that she made an excellent recovery.

A new pattern of ENTEROTRIBE was also shown, which was of great service in shortening and simplifying operations on the intestines in which the bowel was to be occluded as well as divided.

6. *Mr W. J. Stuart* exhibited for *Mr Hodsdon*—

(*a*) A case of HYDATID OF THE KIDNEY which has been successfully treated by incision and drainage. Before the operation, the

blood gave an eosinphibia of 13 per cent., which fell after the operation to 4 per cent.

(*b*) A case of CONTRACTION OF THE PALMAR FASCIA treated by the injection of fibrolysin. The patient, a tinsmith, aged 40, had suffered from the condition, which began in the middle and subsequently affected the ring finger, for twenty years. Before the treatment was begun the middle finger was flexed to less than a right angle, the ring finger to rather more. 20 cc. of fibrolysin were injected every fourth day. After twenty injections combined with the use of a posterior splint the fingers became straight and the thickening in the palm disappeared.

7. *Mr E. Scott Carmichael* exhibited—

(*a*) A little girl æt. 3½ years, from whom four inches of ileum, the cæcum, and ascending colon had been removed for TUBERCULOUS STRICTURE, causing intestinal obstruction at the lower end of ileum.

The interest of the case lay in the early age of the child at the time of operation, which was performed two and a half years ago, the child being at that time 14 months old, extremely emaciated, and weighing 14 lb. 8 oz.

The operation had been performed in two stages, a preliminary ileostomy being followed thirteen days subsequently by secondary resection of the bowel. End to end anastomosis of the ileum to the transverse colon was done, the lumen of the small intestine being somewhat greater than that of the colon, necessitating a somewhat oblique resection of the latter.

(*b*) A boy, æt. 10. A Mongolian idiot previously shown at the Society's meeting after recovery from a GENERALISED STREPTOCOCCAL PERITONITIS, was readmitted to hospital suffering from acute abdominal symptoms two months later.

These were chiefly referred to the upper part of the abdomen and region of the gall bladder. Jaundice was present to a considerable extent.

Operation revealed an empyœma of the gall bladder. This was drained with subsequent complete recovery.

The interest of the case lay in the fact that the child had survived two such serious conditions, and the probability of the gall bladder infection being secondary to the previous generalised peritonitis, the spread of infection having occurred in all probability through the wall of the gall bladder rather than by way of the portal circulation and the bile.

(*c*) A girl, æt. 6, after COMPOUND FRACTURE OF THE SKULL followed by HERNIA CEREBRI. The child had been kicked by a horse while playing on a field, and on admission to hospital was found to have a compound fracture involving the squamous portion of the temporal bone, and the anterior and lower part of the parietal bone, with protrusion and severe laceration of brain substance.

The interest of the case lay in the fact that the child had never lost consciousness and had never had any localising symptoms of cerebral irritation. She made an uninterrupted recovery, in spite of the formation of a large hernia cerebri, which had now almost completely healed.

There was now an opening in the skull about the size of a four-shilling piece.

(*d*) A boy, æt. 6, with CHRONIC ARTHRITIS OF THE WRIST JOINT.

Three years previously a hæmatoma of the thigh was incised. This was followed afterwards by swelling of the right knee-joint, the left ankle-joint, and the first meta-carpo-phalangeal joint. These swellings remained temporarily, and then finally disappeared. A year later swelling of the right wrist-joint occurred, which has persisted, and resisted all treatment, for two years. At different times some of the other joints had been affected temporarily.

There was no evidence of specific disease; Calmette's ophthalmic reaction and the opsonic index to tubercle were negative. Bier's treatment, rest, and massage, tried at different times, had caused no improvement in the condition.

There was no enlargement of the spleen, lymphatic glands, or changes in the blood.

Exploratory incision was made exposing the joint from the dorsal aspect by a radio-carpal incision. The articular cartilages of the scaphoid and semilunar showed small pits or erosions, and the synovial membrane was somewhat thickened.

Microscopic examination showed the condition to be a chronic inflammatory one of non-tuberculous origin.

The pain had disappeared since the operation, and the swelling had improved considerably. It was probably one of those cases of so-called chronic rheumatic arthritis.

8. *Mr C. W. Cathcart* exhibited—

The patient from whom he had removed the right Gasserian ganglion on Dec. 5, 1906. He had shown her previously to the

Society[1] not long after the operation and now that a year had elapsed he thought it well to show her again. She had remained in good health and had no return of pain. Her right eye, however, had become the seat of panophthalmia a few weeks after the operation, and had been removed. Mr Cathcart drew attention to the comparative absence of deformity on the affected side. The zygomatic arch had been removed at the operation, but this diminished rather than increased the deformity; for the atrophy of the temporal and other muscles of mastication due to the operation would have thrown the zygomatic arch into undue relief had it remained in its normal condition.

9. *Dr Norman Walker* exhibited

(*a*) A woman suffering from pemphigus tobaccus. Marked improvement had followed the use of vaccines prepared from organisms isolated from the bullæ.

(*b*) A case of tuberculosis of the skin which had improved after a combined treatment, consisting of the use of small doses of tuberculin at intervals of four weeks, the application of X-rays, and the constant inunction of oleate of mercury.

(*c*) A man, aged 22, showing fine scarring and a patchy eruption over the fore-arms. Although possibly lupus erythematosus, the condition was more probably a very chronic superficial syphilide. Potassium iodide had acted beneficially.

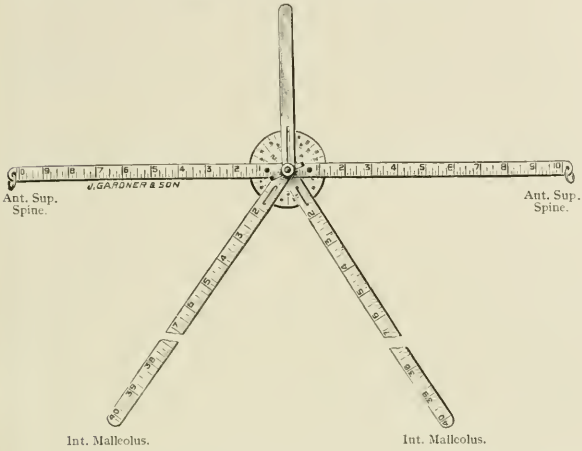
II. Exhibition of Instruments

Professor Chiene, C.B., exhibited—

(*a*) A method of measuring the lower limbs in injury and disease. Pass a string from anterior superior process to anterior spinous process. Take the mid-point and measure from that point to either internal malleolus, at once the difference in the limbs is discovered. Mr Mark Fraser has devised an instrument for the purpose, consisting of a cross piece of metallic tape and two pieces of the same material, both graduated in inches with a dial at the mid-point of the transverse piece of tape to fix the exact angle of the limbs; another strip passed from the mid point up the middle line of the patient's body to prevent any measurement until it is at right angles to the transverse portion. The instrument has been tested in many cases and found most efficient. The late Mr John Duncan

[1] See vol. xxx. page 59.

had an instrument for measuring the angle in cases of hip-joint disease. He called it the Galliometer after one of his students. Professor Chiene has named the instrument recommended the Frasometer, after Dr Mark Fraser, the house surgeon.



(*b*) A mouth guard which hangs free over the mouth and nose. After tying a piece of gauze over the head, a piece of copper wire twenty inches long is tied into the knot below the posterior spinous process; it is passed over the ears and sticks out at either side over the malar bones. A piece of gauze is hung on to the extremities of the copper wire, and it hangs free in front of the nose and mouth. Professor Chiene has found it inconvenient and uncomfortable to tie a piece of gauze over the mouth and nose. By adoption of the method described there is no choking sensation.

(*c*) A spring to be used in flat-foot. It consists of three pieces of metal which are fitted to the foot; the three pieces are united at their extremities. The result is a most elastic bridge on which both the transverse and longitudinal arch of the foot rest. The spring supports both the longitudinal well-known arch, but also the transverse arch across the heads of metatarsal bones. The third side stretches across the transverse arch, which gives way in Morton's disease.

Meeting IV.—January 15, 1908

DR JAMES RITCHIE, *President, in the Chair*

I. ELECTION OF MEMBERS

R. Dods Brown, M.D., M.R.C.P.Ed., D.P.H., Royal Asylum, Edinburgh; Alex. Waddel Greenhorn Clark, M.B., C.M., 24 Braid Crescent; and L. Fourie, M.B., Ch. B., Royal Sick Children's Hospital, were elected Ordinary Members of the Society.

II. EXHIBITION OF PATIENTS

1. *Dr Robert A. Fleming* exhibited—

Case of SPASTIC DIPLEGIA OF INFANTILE ORIGIN. W. F., æt. 43. The patient's birth was normal so far as can be elicited, but from his earliest recollection he was affected as he is to-day. He has spastic paralysis of both arms and legs, but the left arm and right leg are most markedly involved. There are athetoid movements, particularly in the left upper limb, although the other arm and the legs are also affected. In the left arm these movements are mostly flexor-extensor in type, with marked extensor spasm. The movements became much more pronounced on attempting any voluntary effort, and are also increased with mental excitement, but they cease entirely during sleep. When he tries to touch the tip of the nose with his left forefinger, or reaches out to grasp an object, the movements become more and more violent, almost resembling a definite intention tremor in disseminated sclerosis. The right arm and lower limbs, especially the right, show similar but less pronounced movements.

The tendon reflexes are exaggerated in all four limbs, although the muscular rigidity renders it difficult to elicit them in the left arm and right leg, and for a similar reason patellar and ankle clonus are not easily obtained. Plantar extension is present in both legs, but the persistent dorsi-extensor spasm of the right great toe makes it difficult to secure a satisfactory response unless much patience is exercised. The affected muscles are greatly hypertrophied, and the grip with both hands is extremely good.

The patient keeps his left hand in his trouser pocket, as he finds that this device enables him to control the movements most satisfactorily. His gait is spastic, with a hemiplegic outward swing of the right foot.

The back and neck muscles also show marked rigidity, and spasm, mostly tonic, of the splenius capitis, trapezius, and sterno-mastoids is noticeable. There is a curious spasm of the platysma myoides, which becomes evident when he is nervous and tries to speak. The effect is to make him stutter, but this is not so pronounced as when the patient was first seen twenty years ago. He has not got nystagmus. The sensory functions, the superficial reflexes, and the muscular sense impressions are normal. He sweats profusely, but this is largely due to the difficulty he experiences in overcoming the spasm and keeping the movements referred to within check. His organic reflexes are normal. Co-ordination is wonderfully good; notwithstanding the extent of the movements of the left arm, he can with his eyes shut touch the tip of his nose, although he may hit his nose several times in the attempt.

His mental functions are unimpaired. He is fond of books, and both reads a good deal and remembers what he reads. Before he left school he was in the sixth standard. He eats and sleeps well, but he suffers from muscular pains, varying in position, and due apparently to the excessive strain which, owing to his malady, the muscles have to endure. He works intermittently as a French-polisher, being able with his right arm to do some of the coarser work, and he has been in his present situation, with a somewhat indulgent and kindly master, for the past twenty-five years.

There is nothing in the family history of importance excepting that his father, who died of bronchitis at the age of 67, and a brother, who is 38 years of age, both stammered. His mother is 73, and is a delicate woman; one sister died of pulmonary tuberculosis at the age of 30, and his only surviving sister is delicate. There is no history of any nervous affection.

Dr Fleming pointed out that as the birth was natural, and according to the mother's account the patient was born an apparently healthy baby, it was unlikely that the case was one of meningeal hæmorrhage, and that it was probably a meningo-encephalitis, commencing in early life, but with no satisfactory etiological factor. No childish ailment or accident could be assigned as a starting-point of the condition.

2. *Mr J. W. Dowden* exhibited—

A patient after operation for PERFORATED GASTRIC ULCER, showing result of early complete suture of the abdominal wounds.

A. G., aged 35, was admitted to Ward 7 of the Royal Infirmary

with the symptoms of perforated duodenal ulcer. The history was typical of the complaint, and the latter part was of interest from the fact that a physician was consulted some hours before the perforation took place, and instead of milk ordered a light dry diet. Two hours after a meal of fish and toast the ulcer suddenly perforated. The operation was carried out six hours later, when an ulcer on the gastric side of the pylorus and near its upper border was easily found. The ulcer was excised, and the portion removed included a part of the anterior aspect of the pylorus and duodenum. The oval aperture left lay in the long axis of the viscus. Suturing was now carried out, bringing the two ends of the oval aperture together, in fact a gastro-duodenostomy, converting the wound in the transverse axis to a sutured one in the vertical axis. The aperture between the stomach and the duodenum was a very satisfactory one. This method of dealing with duodenal ulcer has proved useful also in two other cases, and is much more rapid than gastro-jejunostomy. A drain was inserted above the pubes and another to the right kidney pouch, and thorough irrigation was carried out with hot saline solution. Fowler's position was adopted in the after treatment and the continuous rectal injection of saline. Thirty hours after the operation it was found that some of the stitches had given way, allowing some coils of the small intestine to escape. An anæsthetic was administered and the intestines replaced, while the healthy appearance of the abdomen warranted the suturing up of the wall in its entirety with excellent results.

3. *Mr F. M. Caird* exhibited—

(*a*) A patient, æt. 50, from whom in October 1907 he had removed half the tongue for epithelioma. The operation was carried out under local anæsthesia with cocaine and adrenaline. The procedure was painless and rendered easy by the help which the patient gave: no gag was required. Immediately after operation the patient spoke without alteration of voice and swallowed readily. Ten days thereafter under chloroform anæsthesia all the connective and lymphatic tissue on the corresponding side of the neck was cleared away from the angle of the jaw to the sterno-clavicular articulation. No trace of malignant disease was found on histological examination of the tissue.

The patient returned in a few weeks, with a hard swelling in the position of the submaxillary salivary gland, which occasionally rose and fell but latterly remained as a firm mass. It was thought to be due to a cirrhosis of the salivary gland and not at

all of malignant nature, which, however, it closely simulated, and the case was shown to elicit the opinion of those present prior to removal of the mass.

The swelling has since been removed, and was found to be purely cirrhotic salivary gland tissue without trace of any epithelioma.

(*b*) A patient, male, who four years ago suffered from acute obstruction due to carcinoma of the sigmoid flexure. For this a simple artificial anus sufficed to save life. A few days later the artificial anus and tumour were removed and the integrity of the colon restored by end-to-end suture. Till recently, the patient had enjoyed perfect health, but he now showed secondary results in the lymphatic glands throughout the abdomen which were inoperable.

4. *Dr Edwin Bramwell* exhibited—

A case which he regarded as very probably an instance of ACUTE POLIENCEPHALOMYELITIS. The patient, a boy aged 9, had had sixteen months previously an attack of vomiting, having up to that time been in the best of health. He continued to vomit at intervals throughout that day. The following morning he complained of pain about the back of the head and neck. A fortnight later he was admitted to the Children's Hospital under Dr John Thomson, through whose kindness Dr Bramwell had an opportunity of examining the case.

The rigidity of the neck and the occipital pain, which was aggravated on movement, were very suggestive of caries of the upper cervical vertebræ. The child left hospital, and extension was applied for several months.

The patient has now no pain, there is no fixation of the neck, although all the movements of the head, notably rotation and forward movement, are markedly impaired. Both the trapezii and sternomastoids are much wasted, and their Faradic excitability reduced. There is probably also weakness of the deep muscles in this region.

The right palpebral fissure is narrower, and the right pupil smaller than the left. This is to be explained by paralysis of the right cervical sympathetic, since the pupil on this side does not dilate either when shaded or when cocaine is introduced into the eye.

The right side of the tongue is atrophic. The palate movement is very slightly defective on the right side.

Dr Logan Turner, who examined the vocal cords, states that the

movements are not impaired. The optic discs are normal, and examination of the other cranial nerves reveals no evidence of disease. There is no weakness of the arms or legs, and no alteration in the reflexes. Calmette's reaction was negative, as was an X-ray examination of the cervical vertebræ.

The points upon which Dr Bramwell based his opinion were the acute onset with vomiting, the implication of certain cranial and spinal nerve nuclei, the purely motor character of the symptoms, and the fact that during the previous six months there had been gradual though slight improvement.

5. *Mr Alexis Thomson* exhibited—

(*a*) A case showing the result after Bier's osteoplastic amputation through the middle of the leg, a portion of the tibia being retained in the anterior flap and brought into contact with the sawn section of the shaft. The operation was more difficult to carry out than an ordinary amputation, but the resulting stump is much stronger and much better able to bear pressure.

(*b*) A boy, aged 17, showing pronounced deformity of the lower limbs from SPASTIC PARALYSIS following on Pott's disease in childhood. There was adduction at both hips so that the thighs crossed one another; the knees were flexed to half a right angle, and the sole of the foot looked directly outwards on both sides. The first stage of the treatment was illustrated on the one side by division of the adductors at the hip, osteotomy of the femur above the lower epiphysis, and transplantation of the insertions of the semimembranosus and biceps into the upper border of the patella. The knee was now extended, capable of movement, and the spastic condition greatly relieved. Further operative treatment was to be carried out in stages.

6. Through the kindness of Sir Thomas Fraser, *Dr Chalmers Watson* was enabled to exhibit two cases of chronic pancreatitis: (1) a man æt. 38, in which the pancreatitis was due to gall-stones, the presence of chronic inflammation of the gland being confirmed at operation; and (2) a man æt. 34, in which the pancreatitis was not associated with gall-stones, but due to an infective inflammation spreading from the duodenum.

III. EXHIBITION OF SPECIMEN

Mr F. M. Caird exhibited—

A specimen of carcinoma of the stomach removed after Billroth's second method.

IV. Original Communication

A HUNDRED CONSECUTIVE OPERATIONS FOR APPENDICITIS: SOME DEDUCTIONS THEREFROM

By J. W. Dowden, F.R.C.S.,
Assistant-Surgeon to the Royal Infirmary

I

The literature on the subject of appendicitis is enormous. Among the resaons for this—apart from the natural interest in the affection—are the various methods of treatment adopted, and the diverse indications for operation which physicians and surgeons of repute have put forward.

That the medical treatment in the acute form of this disease is unsatisfactory can be gauged by the fact that there is no practitioner of experience who, in attendance on a case, can feel certain that he knows what is taking place in the right iliac fossa, and that his treatment will result in recovery. All the time he is oppressed by the uneasy feeling that at any moment serious symptoms may manifest themselves.

The medical man is not the only person who is uneasy about the treatment of this grave disease. The public, alarmed by the many tragic occurrences in well-known families, are asking searching questions, such as, "If the operation had been done earlier, would the patient have had a better chance?" and, "Is the operation such a very serious one, because we have seen deaths announced in the papers as 'following an operation' for appendicitis?"

Some years ago, when my experience was mainly confined to assisting other surgeons in their operations, my interest was strongly aroused in this most treacherous complaint, and the conclusions arrived at then were strongly expressed elsewhere. Strengthened now by an experience of over 100 consecutive operations, my adherence to these conclusions is stronger than before.

It is impossible for me, with the short time at my disposal, to enter into detail, and therefore my remarks will be confined to the main facts which induce me to take up a fixed and definite attitude in the treatment of this disease, confining my statements almost entirely to acute forms of the complaint.

The appendix is usually dependent on a single artery for its blood supply, and consequently unable to cope satisfactorily with the more urgent demands for blood which severe inflammation

calls for. It is impossible to answer the question, "What is the cause of appendicitis?" but in so many of my cases there has been an unclean condition of the mouth that it cannot be ignored as a predisposing factor. Frequently carious teeth were present, and, not uncommonly, enlarged tonsils, and more than one observer has noted the association of tonsillitis with appendicitis. A septic portal to the alimentary canal must sooner or later produce a greater amount of contamination throughout its whole length than is generally the case. As to local factors in the appendix, which predispose to inflammation, the most obvious are concretions and kinks.

Sex as a Predisposing Factor.—In my series of 100 operations 82 were acute cases, and of these 52 were males and 30 females, while the "interval operation" was performed on 14 males and 4 females.

Age as a Factor.—Of the acute cases, 50 were attacked between the ages of 16 and 30 inclusive, 16 were over the age of 30, the oldest being 63, 16 were under the age of 16.

Previous attacks, a fact which is well known, predispose to this inflammation, and in the series before you 37 had had one or more attacks. In one woman 30 years elapsed between her first and second attacks. Deaver, the well-known authority on this subject, says that 75 to 80 per cent. have subsequent attacks.

The pathological classification is very varied, but, clinically, the varieties may be considered as acute or chronic. Those who have been treated for an attack can be divided into those who are cured and those who are not. Those who are cured have had their appendices removed. Those who are uncured retain their appendices, or have gone hence where such things cease from troubling.

Symptoms and Diagnosis.—The average case of appendicitis when seen early exhibits typical symptoms, often spoken of as "cardinal symptoms." There is a history of pain, somewhat sudden in onset, and not infrequently awakening the patient in the early morning. The pain, colic-like in character, is referred to the region of the umbilicus or the epigastrium, and is generally followed by vomiting. It is often on account of these symptoms that the treatment for acute indigestion is prescribed. The pain gradually shifts to the right iliac fossa, where it remains, continuous in type, with, at times, exacerbations. A varying degree of fever is usually present. On examination of the abdomen the right lower quadrant moves imperfectly with respiration, and palpation

in this region elicits tenderness and rigidity, usually most marked at M'Burney's point.

The diagnosis is, as a rule, easy when the patient is seen early, but when the onset is very sudden and severe, or complications have arisen by the time the medical attendant is asked to see the case—not an uncommon event—a definite conclusion is at times impossible, and all that can be determined is the presence of some serious abdominal condition.

The administration of opium renders not only a definite diagnosis difficult, but masks the severity of the attack to a very serious extent. It cannot be too strongly emphasised that every attack of so-called actue or sub-actute indigestion calls for a most careful local examination, including, if there is doubt, a rectal examination as well.

The cardinal symptoms of the disease should be always most prominently kept in mind lest slight deviations should lead the examiner astray.

There is a factor which is apt to delay a diagnosis, and that is the presence of painful spots or areas outside the region of the appendix. The commonest position is to the left and a little below the level of the umbilicus, and on palpation is quite marked. With such a symptom it is helpful to carry out the physical examination in the following manner :—Kneeling on the right side of the patient the right hand palpates the appendix region, inducing pain ; without removing this hand the left should be carefully pressed over the other painful areas, when it will be found that tenderness has disappeared or is very much decreased. If it remains as before there is an appendix passing over to the left side—if the pain is mainly there—or a peritonitis is spreading generally from the pelvis.

In very severe attacks, or where serious complications have rapidly taken place, any abdominal catastrophe may be simulated, and the main point then is not whether to open the abdomen, but where to open it. Three cases so closely resembled perforated ulcer that the duodenum and the stomach were explored first, and later the appendix. Peritonitis was present in all of them, and all of these patients had been under medical treatment for over 24 hours, and been ill for 2 days at least. Another patient was explored over the appendix where peritonitis was found, and the duodenum being examined, through a fresh incision, revealed a perforated duodenal ulcer. Three of these cases recovered.

Two cases of ovarian tumour, with twisted pedicle, closely simulated appendicitis with abscess formation. Both recovered.

A ruptured pyosalpinx exactly simulated appendicitis, but an early diagnosis of severe abdominal lesion by the medical attendant enabled an early operation to be performed, with recovery.

One case was sent to hospital with the diagnosis of suppurative arthritis of the hip-joint. A large appendicular abscess was found of many days' duration extending below Poupart's ligament.

These, of course, are only a few of the conditions which simulate appendicitis.

When a diagnosis of appendicitis has been arrived at, is it possible to gauge the condition of the appendix or its surroundings by the symptoms? The answer to this query is, Not accurately, and, what is more, there is nobody, however skilled, who can tell this with any degree of certainty, or what the termination of the case may be. This is the crux of the whole question of treatment. The local symptoms, the pulse, the temperature, the leucocytosis, even the appearance of the patient—usually so reliable—at best only give an indication of what has taken place, not what is going to occur. These facts must be obvious to all.

Morbid Anatomy.—The most striking point in the morbid anatomy was the rapidity with which changes took place in the appendix and its surroundings. The conditions found at operation bore out absolutely how impossible it is to tell at all accurately the state of the organ from the symptoms.

After-Results and Prognosis.—When appendicitis is diagnosed the prognosis is a very serious one, rendered much more so when pus is present, and of the worst when general peritonitis exists.

That the large majority recover from their first attack is undoubted, but it is impossible at the beginning of an attack to say which case will and which will not.

It can, however, be foretold that a very large number will suffer from subsequent attacks, and it is impossible to prophesy the severity of these later illnesses. Thirty-seven per cent of my cases had previous attacks, 10 of whom required operation on account of the severity of the attack, and 3 of these died. Lastly, it is impossible to insure such patients as first-class lives so long as they retain their appendices. If in a first attack an early diagnosis is made, and an early operation at once proceeded with, the removal of the appendix can be carried out through an incision planned to avoid the possibility of subsequent ventral hernia.

The removal of the appendix is an easy operation as a rule, the acute symptoms rapidly subside, and the patient can be allowed up in a fortnight at the latest, with the guarantee that there will be no subsequent attacks, and a bar to life insurance removed. The prognosis is extremely good. Only 11 patients have been operated on by me at this early stage, and all have recovered, with excellent results. If an abscess has developed in connection with the attack the serious nature of the prognosis is enormously increased, and an operation is performed often with difficulty, and the unsatisfactory feeling that pockets of pus may be passed over, with subsequent ill-results.

The early local dangers are peritonitis, multiple abscesses, obstruction. One case required an anastomosis between the ileum and the ascending colon early in the third week. The general dangers are of course septicæmia and pyæmia.

Late local dangers which arise after operation, when the appendix is left, are the possibility of subsequent attacks. Even if the appendix be removed at the same time as the abscess is dealt with other abscesses may arise even many weeks after the first operation. This complication occurred in two of my suppurative cases. Persistent fæcal fistula may persist. Bands, potential causes of obstruction, may be present, and one case of acute obstruction, which ended in death, was operated on three years after an attack of suppurative appendicitis. Ventral hernia is of comparatively common occurrence.

It need hardly be said that many patients are permanently disabled by ventral hernia or by the exhaustion associated with a long suppurative process, though their lives have been saved. Suppuration was present in 58 of my cases and the mortality was 14. There is scarcely a ward on the surgical side of the Infirmary that has not its case of suppurative appendicitis lying in it.

Treatment.—From my experience I have not the very slightest hesitation in saying that, with our present powers of diagnosis, the only logical life-saving treatment for this disease is the removal of the appendix at the earliest moment.

It is impossible for me to understand how any one who has studied the subject, and its pathology, can for one moment advise anything else. From the merest knowledge it is possible to say that a septic focus of unknown degree in the most intimate relation to the general peritoneal cavity is a most serious condition,

and the only safe treatment is to get rid of it lest other dangers arise.

The surgeon should be called in, not as a last resort, but at the very first; there will be fewer tragedies, and the public will gain more confidence in us than they have already, when appendicitis manifests itself.

It may be said that the patient's relatives were very averse to operation, and so postponement occurred. Every one is averse to operation, but in such cases as these the duty of the medical attendant is to throw the onus of delay on the relatives, otherwise he may be blamed if the termination be a fatal one.

The appendix should be removed, always provided, of course, that it does not entail danger to life, and it will be found that the more experienced the surgeon the more frequently he removes the appendix in suppurative conditions.

It may be said that there are times when a surgeon cannot be obtained, or an interval must elapse before he can operate. In such cases never give morphia. Put the patient on starvation treatment, with enemata of salines after the bowels have been cleared out first by a soap-and-water enema, and locally apply cold or heat, whichever is most comforting.

If a patient recovers from an attack, however mild, he ought to have his appendix removed.

The so-called interval operation with its small mortality is performed on the survivors of acute attacks, and this should be borne in mind.

100 CONSECUTIVE OPERATIONS FOR APPENDICITIS

		Males.	Females.	Mortality
Acute cases	82	52	30	16
Interval cases (survivors of acute attacks)	18	14	4	0
Total,	100	66	34	16

ACUTE CASES

Ages.	Cases.	Mortality.
16 to 30	50	9
Over 30	16	1
Under 16	16	6

Nineteen suffered from previous attacks, and 3 died. Suppuration present in 58 patients. Mortality 14.

	Number.	Mortality.
Operations on 1st day	11	0
,, 2nd ,,	16	2
,, 3rd ,,	18	7
,, 4th ,,	10	1
,, 5th ,,	5	2
,, 6th ,,	1	1
,, 7th ,,	7	2
,, after 7 days	14	1
Total,	82	16

V. Demonstrations

1. *Mr W. J. Stuart* exhibited—

A pneumatic sigmoidoscope. It consists of an ordinary tubular speculum slightly over 30 centimetres in length, with a handle. There is a centimetre scale marked on the outside of the tube, and the end which enters the bowel is rounded and thickened slightly, so as to avoid injury to the bowel. To the observer's end of the speculum there fits on by means of a pivot-and-slot joint a metal collar, to which is attached a fine rod which runs along the inside of the speculum close to the roof, and terminates about 2 or 3 centimetres from the opposite end of the speculum in a tiny but powerful electric lamp. On the metal collar are the necessary electric terminals, and also a small projecting tube communicating with the interior of the collar and therefore of the speculum. Over the projecting part of this metal tube fits the rubber tube of an ordinary rubber ball-bellows, such as is used for Pacquelin's cautery; thus air can be blown into the speculum as desired. In order that the air may not escape, an eyepiece of simple window glass is fixed, also by an air-tight pivot-and-slot joint, to the observer's end of the metal collar. The apparatus is completed by an obturator which fits the speculum when the metal collar and lamp are not in position, and by the necessary conducting cords—with a switch for turning on and off the light—to connect with the accumulator or other source of electric energy. A small rheostat is generally used in the circuit in addition to that which may be on the accumulator, but is not essential.

Method of use.—The patient may be in any position that suits the surgeon, the left lateral position being convenient. The speculum with the obturator is introduced into the rectum, but, as soon as it is well within the sphincter, the obturator is withdrawn,

the lamp-bearing collar and the eyepiece are fitted on and the farther progress upwards of the instrument is carried on under the guidance of the eye. By means of the rubber bellows air is blown in, so that the bowel is distended in advance of the speculum; in this way kinks are unfolded, the friction of the speculum on the bowel wall is practically abolished, while pathological conditions are seen before the tube reaches them. Should the surgeon wish to do something further, for example, to remove a portion of a growth for examination or to pass a bougie through a stricture he gets the tumour or the stricture opening opposite the end of the speculum, removes the eyepiece, thus allowing the air to escape and the desired portion of bowel falls across the mouth of the speculum. Before withdrawing the speculum, any air should be allowed to escape, although the actual amount introduced is not great.

Advantages.—A number of the disadvantages of the older simple sigmoidoscope, such as that of Kelly, are removed by the use of this newer pattern of instrument; *e.g.*—

(1) Kelly's speculum depended for the entrance of air into the bowel upon negative pressure inside, caused by the abdominal contents falling towards the upper part of the abdomen. Hence the patient had to assume a knee-shoulder attitude, a position uncomfortable for any one and particularly disagreeable to female patients. With the pneumatic speculum air is *pumped* in, and therefore the patient may be in any position.

(2) The light was thrown in from without, either by a forehead lamp or by a forehead mirror. This was often awkward for the surgeon, and in any case involved great waste of time if several people wished to see the condition. In the pneumatic sigmoidoscope, the lamp being inside, an observer has merely to apply his eye to the speculum.

(3) As the older instrument was passed by touch and was attended by considerable friction upon the bowel wall the latter, especially if ulcerated or otherwise pathological, might be damaged. With the pneumatic sigmoidoscope this risk should not exist, as it is passed under the control of the eye, while the inflation of the bowel reduces friction to a minimum.

It seems not unreasonable to assume that the pneumatic sigmoidoscope will soon entirely displace, as it has already considerably displaced, the simple tube which was previously employed

2. *Dr Chalmers Watson* gave a demonstration of the end reaction

of the recently devised PANCREATIC REACTION IN THE URINE (Cammidge), illustrated by lantern slides, showing the characteristic crystals of phenyl pentose azone.

3. *Dr Robert A. Fleming* gave a LANTERN DEMONSTRATION OF CHANGES IN THE OPTIC NERVES IN CASES OF INTRACRANIAL TUMOUR. Dr Fleming showed the neuroglial elements in a normal optic nerve, and demonstrated the mesoglial cells which are not distinguishable by ordinary methods of staining, but which may be differentiated by Dr Ford Robertson's platinum method of staining. In optic neuritis, however, these mesoglial elements are apparently unaffected unless it be that they increase slightly in number, and the neuroglial elements alone show the characteristic proliferation and hypertrophy which he demonstrated in sections from a series of cases. The hypertrophy much resembles a condensation of cell processes, and the greatly enlarged cell lacks a definite outline in many instances. The nucleus is eccentric in position (as it usually is in the normal neuroglial cell), but is much enlarged, stains less deeply with nuclear stains such as hæmatoxylin or methyl blue, and definite mitotic figures can be seen. Some of the larger cells show what appears to be vacuolation. These hypertrophied neuroglial cells are most obvious near the periphery of the nerve, and do not extend continuously throughout the chiasma and optic tracts to the brain, but are most definite near the eyeball and between the eyeball and the entrance of the central artery and vein of the retina into the nerve, although they may be found centrally as far as the optic foramen.

In early optic neuritis the change may be seen, but it reaches its most marked stage when optic atrophy commences.

Dr Fleming showed that similar proliferation and very marked hypertrophy of the neuroglial cells was found near the tumour nodules in the brain in three cases in which he had obtained specimens, but that the changes in these cells was not continuous from tumour nodules along optic tracts to the nerves. He showed that normally there were fewer neuroglial elements in the optic tracts and chiasma, than in the orbital portion of the optic nerves.

A series of optic-nerve sections were shown from cases of albuminuric retinitis in which neuroglial changes were demonstrable similar to those in optic neuritis due to tumour.

In contrast, optic nerve sections from cases of tubercular meningitis and other forms of meningitis were next demonstrated, and in these no such neurolgial hypenrtrophy and proliferation was seen.

Dr Fleming supported the view enunciated first by Leber, that the neuroglial changes were of toxic origin, and that they were primary. The toxine in intracranial tumour cases and in cases of albuminuric retinitis acts on nerve fibres and neuroglial cells, and the neuroglial hypertrophy and proliferation is not merely secondary to the action of the toxine on the nerve elements. The neuroglial changes are closely related as regards position to the blood-vessels and lymph channels, and it seems probable that the toxic agent is conveyed by them.

He pointed out that in cases of tubercular meningitis the incidence of optic neuritis depended on the co-existence of one or more tubercular tumours in the brain, and he showed in other cases of meningitis due to the pneumococcus, etc., that there was generally a leucocytic invasion of the optic nerve, but no neuroglial proliferation or hypertrophy.

Cases of intracranial tumour in which there was no optic neuritis were referred to, and the difficulty of accepting the toxic theory was discussed; but the absence of the neuroglial proliferation and hypertrophy in cases of optic atrophy not of the consecutive type, and of which several illustrative slides were shown, rendered the view that the neuroglial changes were purely secondary to optic nerve fibre destruction absolutely impossible of acceptance.

Dr Fleming in conclusion showed how clearly in the cases of marked optic neuritis going on to consecutive atrophy, the dual nature of the neuroglia was demonstrated, the mesoglial elements showing no hypertrophy and very doubtful increase in numbers, while the neuroglial cells proper alone exhibited definite hypertrophy and proliferation.

Meeting V. February 5, 1908

DR GEORGE HUNTER, *Vice-President, in the Chair.*

1. EXHIBITION OF PATIENTS

Mr F. M. Caird again exhibited the male patient, aged 50, presented at last meeting of the Society. At that time after removal of half the tongue and all the lymphatic tissue on the corresponding side of the neck, there had appeared a hard rounded mass under the jaw, which was diagnosed as non-malignant, and due to a simple inflammatory cirrhosis of the submaxillary salivary gland.

Removal of this mass had now been effected, and corroborated the diagnosis, since not a trace of epithelioma had been discovered, and the condition was exactly as anticipated. The patient was in good health.

II. Exhibition of Specimens

1. *Mr Scot Skirving, C.M.G.*, exhibited—

A large pedunculated fibroid, uterus, and annexa removed from a patient aged 70, in whom a complete twist of the *uterus* had occurred, producing strangulation of the uterus and symptoms of acute intestinal obstruction.

The specimen was shown on account of its great rarity. The patient, a married woman who had never been pregnant, had suffered from occasional attacks of pain in the abdomen during the last two years, but until a week before admission to Leith Hospital had never had any symptoms of intestinal obstruction. On this occasion, however, in addition to pretty acute abdominal pain, sickness and occasional vomiting, absolute constipation for some days, and definite (although not extreme) distension were present.

On admission, a large, hard tumour could be made out, occupying a considerable portion of the abdominal cavity, especially on the right side. A striking feature of the case, also, was excessive vaginal tenderness, which made all examination in this region impossible. Under anæsthesia, the tumour was found to be capable of being moved from side to side, and to consist of two masses apparently connected with one another.

On opening the abdomen, it was seen that the uterus was much enlarged, and completely twisted on itself at the junction of the cervix with the body, so that the posterior surface looked directly forwards. The congestion was so extreme that the broad ligaments and ovaries resembled an overdone mushroom in colour. Several small pedunculated fibroids were present in addition to the large bilobed one referred to above. The latter was connected with the uterus by a pedicle nearly as thick as the wrist, and did not show much congestion. It had, apparently, shared in the general rotation, and its pedicle, therefore, was not twisted. There were no adhesions.

Supra-vaginal hysterectomy was rapidly performed (in twenty minutes). The patient did well for five days, and seemed likely to make a good recovery. On the sixth day, however, hypostatic congestion appeared, and she died on the seventh day.

The intestinal symptoms were probably reflex, as no definite evidence of pressure on any portion of the intestine was found at the operation

The whole specimen weighed six pounds ten ounces.

2. *Dr Michael Dewar* exhibited—

A dissecting ANEURISM OF THE ASCENDING AORTA with a transverse rupture about one inch above the aortic valves. The patient, a man 52 years of age, was in apparent good health. There had been no previous signs or symptoms of cardiac or aortic disease. On returning home from business on the evening of 6th January, he had a slight fall on the slippery street, from which he experienced no inconvenience. After a light supper he was engaged in examining and writing an article on "Church Tokens" till midnight, when he retired to bed. He was suddenly seized with intense pain over the manubrium sterni about 1.30 A.M., without dyspnœa in the slightest. He received a hypodermic injection of $\frac{1}{2}$ gr. morphia at 2.30 A.M. This did not ease the pain. At 3.30 A.M. a second injection of $\frac{1}{4}$ gr. morphia was used, after which he slept for one hour. The pain was still intense, but no dyspnœa. The pulse was going fairly strong and quite regular. During the early morning his face became pinched and somewhat pallid, and several attempts at evacuation of the bowels were made.

Shortly after 7 A.M. a sudden collapse took place, and the pulse became almost imperceptible. The $\frac{1}{60}$ gr. of strychnine was injected at 7.30 A.M. A consultation was then held, and while a soap-and-water injection per rectum was being administered, he, while lying on his right side, suddenly expired at 8.45 A.M.

At the post-mortem, the lungs were found healthy with a small amount of pleural effusion. The pericardial cavity was full of dark blood and soft clots. The heart was slightly larger than usual, but firm and healthy. There were no adhesions, and the valves were competent. On examining the aorta a transverse rupture about 1¾ inch in length was seen in the thinned wall, involving the three coats, with blood clot surrounding the external coat. There was no sac. The sequence of events was evidently the following—

An undiagnosed dissecting aneurism—after the fall the thinned coat gradually gave way, blood at first oozing into the pericardium—rupture suddenly extended after 7 A.M. with large effusion of blood into the cavity, at which time the pulse became almost imperceptible, the patient living fully an hour and a half afterwards.

The case is interesting, owing to the absence of previous sign and symptom, and its rarity.

III. Original Communications

1. ELECTROLYSIS IN MEDICINE AND SURGERY

By Dawson Turner, M.D., F.R.C.P.Ed., Lecturer on Physics, Surgeons' Hall

Ten years ago electrolysis was chiefly practised for its destructive or coagulating power, *e.g.*, for nævi, removal of hairs, goitre, aneurisms, and strictures. Since that time the method has been much extended and it is now used to introduce medicaments through the skin into the tissues by means of ions. Among the leaders of this new development are Edison, Leduc, and his pupils, Gautier, etc. Fifty years ago Benjamin W. Richardson had, however, endeavoured to produce local anæsthesia by the electrolytic introduction of aconite. Any attempts that were made in this direction in former times were classed generally under the heading cataphoresis, by which a bodily transfer of the electrolyte, usually in the direction of the current, was meant, but the ionic migration of the ions is of far more importance. Some knowledge of the physics of the process is required; only those medicaments which behave as electrolytes (which break up into ions) can be so introduced; *e.g.*, aqueous solutions of salts, acids, and bases. The particles of such a solution are believed to be more or less dissociated according to the dilution, and the electric current is carried through such solutions by the bodily movement of the ions; it is a convection current of electricity. Now the human body is an electrolyte, and electrical currents are only conveyed through it by the movements of ions; the body consists of various tissues bathed with an alkaline medium and surrounded by an insulator, the skin. The resistance of the skin can be diminished by thoroughly moistening it with an electrolyte so as to afford an abundance of ions to carry the current, and by increasing the area of contact of the electrodes. The smaller the area of contact of the electrodes, the greater will be the current density, and the greater the discomfort felt by the patient. Suppose a person to place each hand in a separate basin of salt water, and suppose these are connected with the terminals of a source of electric supply, such as a battery or switchboard connected to the main, then at the anode or basin connected to the + pole, the body will lose its anions and receive cations, while at the cathode or basin connected to the − pole, the body will lose its cations and receive

the anions. The anions are constituted by the acids radicals or elements, and the cations by the metallic radicals or metals. Thus oxygen, chlorine, sulphion, and hydroxyl are anions, while hydrogen, sodium, copper are cations. When sodium chloride is dissolved in water, its molecules dissociate into ions or particles with opposite electrical charges; the anions are charged −, and will therefore be attracted to the anode, and the cations are charged +, and will be attracted to the − pole or cathode. Now in this case, Na + is the cation and Cl − is the anion. At the anode the body will yield up chlorine and receive sodium, while at the cathode it will give up sodium and receive chlorine, for we may regard the tissues as principally bathed with NaCl. Between each electrode there will be a migration of ions; the Cl − ions will be moving to the anode and the Na + ions to the cathode. If a piece of raw beef be taken and electrodes be applied to either side of it with some blotting-paper soaked in a solution of KI under the − electrode, and some blotting-paper soaked in starch, between the − electrode and the beef, then if a current be passed, it will be found that the blotting-paper at the + pole will shortly turn blue, showing that the iodine has been carried from the cathode to the anode through the beef. In one of the writer's experiments the blue reaction was obtained in twenty minutes when the beef was ½ inch thick, and a current of 50 m.a. was maintained. Experiments have also been made on living beings. Thus Edison (Int. Med. Congress, 1800) placed one hand of a man in a solution of lithia and the other in a solution of soda, on a current of 4 m.a. being passed for eleven hours; spectroscopic examination of the urine showed that considerable quantities of lithia had been absorbed. Leduc of Nantes has performed some instructive experiments: he connected two rabbits by a pad of moist lint and applied an electrode wetted with strychnia sulphate to each of them. On passing the current the rabbit at the anode was poisoned by the strychnia cations passing in, while the rabbit at the cathode, which received only the anion So_4, was unaffected. If, however, the electrodes were moistened with potassium cyanide, then the rabbit at the cathode was poisoned through the migration of the anion cyanogen. Leduc also experimented on himself and produced toxic effects by the ionic introduction of morphia and strychnia. These are then sufficient proofs if any were needed, that substances can be passed through the skin by electrolysis. It remains to consider to what useful purpose this process can be put. The following is a list of some of the con

ditions for which electrolysis has been, or may be advantageously applied: Rodent ulcer, parasitic affections, sinus and fistula, anchylosis, sclerosis, fibrous adhesions, strictures, tic-douloureux, neuralgia, sciatica, rheumatic and gouty conditions. Leduc was one of the first to treat rodent ulcer by the introduction of the zinc ion; he reported the complete cure of a case by one application. As a rule, more than one application is necessary. The method is as follows:—

Having as far as possible cleaned the surface of the ulcer, apply to it eight or ten layers of lint cut a very little larger than the size of the ulcer; soak the lint first in a 5 per cent. solution of zinc sulphate; over the lint place an electrode, if possible of zinc, to avoid the introduction of other ions; connect this to the + pole, and connect the — pole to a basin of salt water into which the patient may place his hand. Raise the current very gradually to as much as the patient can conveniently bear (40-60 m.a.), and maintain it steadily for thirty minutes, then gradually diminish it to zero. The surface of the ulcer will now be found to be whitened and dry. If any part has escaped the action, a fresh application limited to that part should be made. The patient should be seen again in a week or fortnight, when another application can if necessary be made. Many cases have been treated successfully in this way in the electrical department of the Royal Infirmary. The following are notes of a case.

D. H., aged 61, recommended by Dr R. Maclaren, June 6th, 1907. History—Six months ago a wart appeared on the right side of the nose; this grew steadily in spite of various applications. On admission the growth measured $\frac{3}{4}$ inch across, and its margins were raised and hard. It was photographed and then treated with zinc electrolysis, a current of 40 m.a. being passed for thirty minutes. When seen again a fortnight later, the ulcer was much improved, and the lower hard margin had melted away. A week later a second application was made. On July 3rd, he was exhibited to the Medico-Chirurgical Society. September 16th, returned practically well; was photographed again. All cases of rodent ulcer do not do so well; old ones with a large surface or those which have ulcerated into cavities may not yield to this treatment. Chronic ulcers, sinus and fistulæ, are also amenable to the zinc ion. A sinus should be plugged with a shred of lint moistened with zinc sulphate solution, and a zinc wire should pass into the sinus so as to make contact with the lint; this should be connected to the + pole. One application may sometimes suffice. The copper ion can be tried in ringworm and

sycosis. Leduc has found that in fibrous anchylosis and cicatricial contractions the chlorine ion has a loosening and resolving action; this has been confirmed by the writer in several cases. A. B., under the care of Mr Cotterill, a case of anchylosis of the left knee patient met with an accident ten months ago; this resulted in a septic condition of the knee into which many incisions were made, and the patient was in bed for fifteen weeks; the knee became stiff and could not be flexed to more than a right angle. The patient was now treated with chlorine electrolysis, and every application resulted in greater freedom of motion. After three applications his foot could move through an arc of 7 inches more. A case of Dupuytren's contraction fingers was treated in the same way the forefinger was bent to a right angle, and the disease had lasted fifteen years. Each treatment loosened and eased the contraction, and after eight applications of half an hour each the finger could be straightened. But sclerosis of other tissues, provided they are not too far removed from the surface, can be successfully attacked. In this way, the writer accounts for the great improvement he has obtained in cases of spastic paralysis, by passing very strong currents with one electrode moistened with sodium chloride over the affected part of the cord and the other over an indifferent part. Some part of the current will traverse the affected segment, there will be an exchange of ions, and the action of the chlorine ion will have a resolving effect on the sclerotic tissue, so that nerve impulses will be transmitted more freely. Instead of the chlorine ion the hydroxyl ion OH can be used. Leduc has cured the most rebellious strictures of the urethra by passing a metallic bougie attached to the − pole up to the contracted part, and by passing a small current for ten minutes twice a week. Similar treatment can be employed in strictures of the cervix, œsophagus, etc. For cases of tic-douloureux, the salicylic ion is useful. The area of distribution of the affected nerve should be covered with several layers of lint soaked in a weak solution of sodium salicylate. An electrode attached to the − pole should be placed over this, and a large electrode attached to the + pole over the cervical vertebræ. Very great care must be taken to avoid all abrupt changes in the current strength, or the patient will be rendered giddy and will see flashes of light. The current should be gradually raised to a point short of discomfort to the patient (15-40 m.a.), and should be maintained for thirty minutes. The following case is instructive.

R. M., aged 58, recommended by Dr Boyd; patient had suffered

from tic-douloureux for five months; the pain was stabbing and worst at night, but never away. Patient unable to sleep and up and down all night; cannot open jaw; becoming worn out and exhausted; many remedies tried unavailingly. The first application of 30 m.a. for twenty minutes relieved him for twenty-four hours; a second application three days later had a similar effect, after five applications the patient expressed himself as being greatly benefited; he was able to take food and to sleep, and the attacks had almost ceased. Sciatica and pleurodynia can be similarly relieved. Another class of cases in which the method may be of service is that of local affections, or where there are local manifestations of some general disorder—in gout, rheumatoid arthritis, and chronic rheumatism and syphilis. A gouty joint can be directly attacked by placing it in a bath containing a solution of salt of lithia connected to the + pole; on passing a current the lithia will be carried into the affected part and uric acid and oxalic acid, etc., will tend to pass out. Joints affected with rheumatoid arthritis or chronic rheumatism can be treated according to the same method, but other drugs, such as sodium salicylate, may be found more useful. The writer's results with cases of rheumatoid arthritis treated with the salicylic ion have not been very encouraging. Much may be expected from the electrolytic introduction of drugs in the future; instead of having as in former times to give a patient relatively enormous doses by the stomach, which may be injurious to the digestion or toxic, in order that a distant joint or locus morbi may receive a sufficient supply, we can without disturbing his general condition introduce directly into the affected part the drug that is likely to prove most useful. There are, however, certain limitations: one is that all drugs cannot be so introduced, only those that dissociate into ions. For example, oils, alcohols, glycerines, do not form ions and do not conduct electricity; the spiritus chloroformi and the glycerinum acidi carbolici could not be used in electrolysis. To introduce these substances electrolytically their aqueous solutions must be taken. For this reason too they are more toxic in aqueous solution when taken internally. Electrolytes also differ in their ionisation, acetic acid and ammonium hydroxide are but slightly ionised and are therefore poor conductors and more difficult to introduce by this method. Dr Gonzales Sanchez has made in Prof. Leduc's laboratory a study of the behaviour of different ions and of the reaction of the skin and tissues to their electrolytic introduction. I quote some of his results.

The halogens and alkalis are easily introduced and produce, in dilute solution, no untoward effects. The alkali earths are also well borne, but if pushed may produce necrosis. The introduction of the ion magnesium is painful, a rose colouration of the skin is produced, traces of which can be seen for more than fourteen days later. Manganese also produces a punctiform dark colouration which may last a couple of months; the dark points indicate the position of the glands, and, as Leduc has pointed out, are a proof that the current enters only by the glands. The ions of the heavy metals coagulate albumen and are caustic. How far any ion can be made to travel in the tissues is a matter of great interest; upon this depends the value of the method in deep-seated affections. Much must depend upon the length of time the current is maintained, the current strength, the velocity of migration of the particular ion, and its diffusibility. There seems to the writer to be no reason why in the future it may not be possible to attack a phthisical cavity directly. Surely this would be a great step in advance.

2. REMARKS ON EMPYEMA, BASED ON A SERIES OF ONE HUNDRED CONSECUTIVE CASES

By Alfred E. Morison, M.B., F.R.C.S.Ed., Hon. Surgeon, Hartlepools Hospital

In considering empyema there are one or two points to which I should like to draw your attention.

I. The Physics of Effusion into the Pleural Cavity.—In the normal condition of the pleura the intrapleural pressure is negative, but becomes positive in purulent effusions. Effusion into the pleural cavity *first shows itself* as an area of dulness—(*a*) Just below the angle of the scapula. (*b*) In the axilla between the fifth and eighth ribs. (*c*) In obliteration of Traube's semilunar tympanitic space. (*d*) In the right front near the angle made by the cardiac and hepatic lines of dulness.

Disappearing effusion is last manifest in the axillary line between the fifth and eighth ribs.

The root of the lung is its fixed point, and with increasing effusion the lung becomes compressed round it. The re-expanding lung last touches the chest wall at a point diagonally furthest from its fixed point, *i.e.* in the axilla about the fifth interspace. This is important to bear in mind, as it is the point of election, in cases of general purulent effusion for incision and drainage. It also

explains why emptying of the pleural cavity in " empyema necessitatis " occurs generally about the fifth interspace.

The lung does not tend to collapse on its fixed point when air is admitted into the pleural cavity. The intrapulmonary pressure is quite sufficient to maintain the two layers of pleura in contact except for a small area round the point of entrance of the air.

II. Interlobar Fissures.—The fissures dividing the lungs into lobes can be mapped out as follows :—A line drawn from the spine of the second dorsal vertebra downwards and forwards to the junction of the sixth costal cartilage with the rib divides the lower lobe from the upper ; all above and in front of this line is upper lobe, all below and behind this line is lower lobe. On the right side a second fissure extends from the middle of this line just named to the junction of the fourth costal cartilage with the sternum. These fissures or sulci vary in depth, that between the upper and the lower lobes being deeper, and extend almost to the root of the lung. Pus forming in this fissure is very apt to escape detection, being transverse to the axis of the body, and very commonly is the form of empyema which empties itself through a bronchus.

III. Causes of Empyema.—1. *Pleuro-pneumonia*, as Dr Drummond of Newcastle first pointed out, is by far the most frequent cause of empyema. In 91 per cent. of my cases the history left no doubt of the truth of this important observation. The rich network of lymphatics immediately under the pulmonary pleura, the invariable association of pleurisy with pneumonia, the constant presence of the pneumococcus in pneumonic lungs, and a recognition of the fact that the pneumococcus is capable of causing suppuration, prepare one to receive an explanation supported by experience and the teachings of anatomy and physiology.

2. *Pleuritis.*—This is by no means so common a cause of empyema as is generally supposed. Only 4 per cent. of my cases appeared to be probably due to this cause. The number of ordinary cases of pleuritic effusion seen which never suppurate is remarkable if this be a frequent result. If an ordinary serous effusion becomes purulent, a dirty needle or aspirator is the cause in the majority of cases—some would say always.

3. *Tuberculous disease of the lungs*, with signs of cavity, occasioned empyema in two of my cases (2 per cent.). The purulent pleurisy followed a pneumothorax, as is the usual course in tuberculous cases.

4. *Tuberculous disease of the lower lobe of one lung, without signs of cavity*, was followed by empyema in one patient

Case 1.—G. B., æt 43, has had a cavity at his left apex for some years. Seven weeks ago he had an attack of acute pneumonia at his left base. His present complaints are a cough with profuse expectoration, shortness of breath and loss of flesh. From being a stout man he has been reduced to a very thin one. The cough, he says, comes on at intervals, and at these times a large quantity of yellow expectoration is expelled by it. Relief is obtained for a time; then the cough and expectoration are repeated. Physical examination shows an apical cavity apparently about 4 in. in length. Posteriorly and laterally the left base up to the lower angle of the scapula is dull on percussion, the vocal fremitus is unaltered, and crepitation and increased vocal resonance are heard over the dull area. An aspirator needle introduced between the sixth and seventh ribs posteriorly, proved the presence of pus. The chest was incised at this spot, and 9 oz. of pus escaped. The immediate result was most satisfactory, all his symptoms being relieved. The tube was removed in three months, and the sinus closed, but crepitation continued still at the left base.

5. *Septic disease of abdominal organs* may produce empyema directly, by direct continuity of tissue or by means of the diaphragmatic or retroperitoneal lymphatics, or, indirectly, by setting up a septic pneumonia. It is not an infrequent complication in abscess of the liver, appendicitis, perforating gastric or duodenal ulcer, acute pancreatitis, etc. Such a case was the following:—

Case 2.—A. M'G., æt. 30, was seen with Dr Boswell on 10th January at 11 A.M. complaining of severe pain in the epigastric region of twenty-seven hours' duration. He gave the following history:—On the previous morning at about 8.30, while walking along the street, he was seized with violent pain in the pit of the stomach. He vomited immediately, but this gave him no relief. With difficulty he was able to return home a distance of about 300 yards, and at once went to bed. Dr Boswell saw him twenty-four hours later, and, recognising the serious nature of the illness, asked me to see him. He gave a past history of having had some slight indigestion during the previous three weeks and of feeling depressed and tired, but beyond this he ailed nothing.

On examination, we found him looking ill, with an anxious expression; temperature 100°, pulse 92, respirations, 32

Breathing was almost entirely thoracic, the abdominal msucles were rigid in the epigastric region, and there was great localised tenderness on pressure under the right rectus muscle. Vomiting occurred as soon as anything was taken.

The diagnosis we made was a slight leakage from a duodenal ulcer, but, taking into consideration the length of time that had already elapsed, we administered a $\frac{1}{4}$ gr. of morphia hypodermically, and decided to open the abdomen in two hours if there were no improvement in his condition. On visiting him at 2 P.M. he was very much better, had not vomited, had slept most of the time, the acute pain had disappeared, and there was only some tenderness on deep pressure in the right hypochondrium. He was ordered nutrient enemata, and forbidden to take anything by the mouth. He progressed satisfactorily, except that his temperature remained raised from 99° to 102°, until the 21st, when he began to be troubled with pain in the right side of the chest and a short dry cough. On examining his chest, we found friction at the base of the right lung posteriorly, and dulness merging into the liver region was elicited on percussion. As the dulness extended, the breath sounds at the base of the lung became diminished, and on the 24th an exploring needle inserted into the pleural cavity through the eighth interspace posteriorly, drew off some stinking sero-purulent fluid which had a strong acid reaction. On the 25th I opened the right pleura in the eighth interspace posteriorly, when about a pint of fluid, similar to that withdrawn by the exploring needle on the previous day, was evacuated. His temperature at once fell, and remained below 100° except on 1st February, when it rose to 101°. This was found to be due to blocking of the drainage tube by a large slough. On removing this his temperature fell to normal. The tube was removed on the 12th, and after this date the patient convalesced rapidly, and was able to leave home on the 24th with the wound healed.

6. *Staphylococcic or streptococcic infection of the lung, etc.*—One such case has come under my notice due to an osteo-myelitis of the ribs.

CASE 3.—B. B., æt. 17, consulted me on 16th August 1905. Her mother noticed that for about six weeks she had been very short of breath, was very easily tired, and had become very much thinner. On examination, I found her temperature 101°, pulse 116, and feeble; she had an ashen complexion, and looked very ill. On inspection of the chest, the heart was seen beating under the right breast,

movement on the left side was practically nil, and there was a general fulness between the ribs. On percussion, the whole of the left chest was absolutely dull; breath sounds were entirely absent, and the heart sounds were most distinct on the right side. On introducing an exploring needle into the left chest, thick pus was withdrawn.

August 18.—Operation.—The left pleural cavity was opened in the fifth interspace in the mid-axillary line, and about 3 pints of thick pus escaped. A large drainage tube was inserted. After this she rapidly improved, the discharge, at first very profuse, gradually diminished, but a large cavity remained in the pleura. On 6th November chloroform was administered, and a thorough examination made. The whole of the fifth rib was found to be necrosed and removed. On 2nd December the wound was again examined under chloroform. The lung was still unexpanded, and the sixth rib was felt to be bare and removed. A counter-opening was also made in the seventh interspace posteriorly, and a second drainage tube inserted. Considerable improvement took place in the patient's condition after this until 17th December, when she began to complain of pain and stiffness in the back of the neck. This gradually became worse, her temperature ran up to 104°, sickness set in, and she became comatose, and died on the 20th of cerebro-spinal meningitis. Unfortunately no post-mortem could be obtained.

Diagnosis.—In the ordinary case, the patient is being attended for pneumonia, and presents the usual symptoms and signs of that disease. The acute illness runs its course, and convalescence seems assured. Then it is noticed that the patient does not recover as was expected. He becomes listless, has a poor and fitful appetite, loses flesh, and sweats at nights. Vomiting is a not infrequent symptom, and has appeared to me more common when the right side is affected. There is a short dry, troublesome cough, and a febrile temperature. A peculiar ashiness of complexion may be observed. With symptoms of this kind, a history such as has been described, and physical signs of fluid in the pleura or of consolidated lung, the exploring needle should find pus.

In several cases I have seen the diagnosis has been difficult from the misleading character of the history and symptoms, and has only been arrived at by a complete examination and confidence in physical signs. The difficulty arises from the curious simulation of an abdominal ailment by certain of these cases. The patient, if a child, complains of pain in his "belly." The illness commences acutely with vomiting, distension, tenderness, and hardness of the

abdomen. The cough is not an aggressive feature, and it is easy to regard the case as one of peritonitis, which it closely resembles. In some cases of simple pleurisy, the same abdominal tenderness, resistance, and pain are seen, and the explanation is obvious if the distribution of the intercostal nerves is remembered, the sixth terminating in the epigastric region, the tenth in the umbilical region. The pleuritic pain is referred to the end of the irritated nerves; the skin supplied by them is tender, and the abdominal muscles enervated by them are contracted. When, too, it is remembered that acute idiopathic peritonitis is one of the rarest of ailments, such a mistake is not likely to happen. The physical signs are those of fluid in the pleura, deficient expansion of the affected side of the chest, and approximation of the ribs, with diminution in size of the intercostal spaces, is the rule, and it is only in rapidly forming effusions that separation of the ribs with bulging of the intercostal spaces and œdema of the chest wall occurs.

Absolute dulness and resistance on percussion are present, it may be over practically the whole side or over a limited area and in any part of the lung.

Vocal fremitus is weak or absent, and its localisation is sometimos difficult to test accurately. The old-fashioned single stethoscope will be found valuable as an aid. By applying the ear-piece to the palm of the hand and the chest-piece to the suspected site, small localised areas of altered fremitus may be mapped out.

Vocal resonance is also of great assistance, and corresponds with the condition of the breath sounds. When the latter are absent, voice sounds are also absent; when the breathing is tubular, the vocal resonance, and especially a whisper, is intensified. In children who have thin chest walls it is specially unreliable. Bacelli's theory—that the whispered voice is conducted through serum but not through pus—has not in my experience proved of any value as an aid to diagnosis. The nature of the breath sounds depends on the sounds that are produced in the lungs or in the bronchi, and not upon the presence of fluid. Fluid transmits sound well, but there may be no breath sounds produced, and so none are audible through the fluid. Tubular or bronchial breathing is present in a fair proportion of cases, and, especially in children, owing to the greater resiliency of their chest wall, a correspondingly small degree of compression of the lung, and greater patency of the bronchi.

The heart is frequently altered in position, and the heart sounds are absent from their normal position, or present in some other

region, such as the left axilla or to the right of the sternum. In some cases the side is hyper-resonant or the percussion note little altered, especially when the effusion is on the left side. In such a case the tympanitic area of resonance usually occupied by the stomach laterally will be dull, and the dulness extends over as far as the epigastric region. These signs are due to the diaphragm being pushed down by the fluid in the pleural cavity, and displacing downwards the abdominal contents. On the right side the dulness of fluid in the chest may be mistaken for the normal liver dulness, if the lower edge of the displaced liver is not recognised.

In all doubtful cases the exploring needle should be used. It may be employed with perfect safety, if the skin round and over the proposed puncture be previously purified and the needle rendered aseptic.

In some cases several punctures are necessary before the result is certain, and even though the needle goes into pus this may fail to be withdrawn. In every case where nothing comes through the needle, it should be suspected of being blocked. These difficulties arise in localised empyema, or where the pus is inspissated or flaky.

Prognosis.—1. That pus in the pleural cavity is capable of *absorption* has been demonstrated to me by one case which came under my notice—A. S., æt. 3. The presence of pus was diagnosed by physical signs and verified by the needle, but the parents refused operation. Recovery followed without expectoration or external discharge of the pus. In another case, C. W., æt. 4, operative interference was refused for six weeks after the diagnosis of empyema was made by the needle, and on opening the chest only 4 oz. of thick sticky pus came away. It looked as if the pus were in process of absorption.

2. *Spontaneous evacuation through a bronchus.*—In two cases recovery followed this event without operation. C. W., æt. 6, had a large empyema on the right side. Operation was refused. The pus, ruptured into a bronchus, was expectorated freely, and recovery took place in twenty-one days. R. U., æt. 49, had pneumonia of the middle lobe of the right lung on 3rd August 1905. Interlobar empyema discovered by physical signs and exploring needle on 26th August. Operation refused. Pus was evacuated by expectoration until 24th September. Complete recovery.

In ten cases pus in such large quantity as to make it certain that an opening had formed in the lung was expectorated prior to operation. After operation these cases pursued an aseptic

course, and complete recovery without damage to the lung followed in all. Most of them came to me with a diagnosis of consumption.

3. In some cases *pus* is discharged into the *œsophagus, stomach,* or other adjacent organs. This can only take place where the accumulation is great and the course of the disease is prolonged and unaided. I have no experience bearing on it.

4. *Spontaneous evacuation through the chest wall,* the so-called "empyema necessitatis," is of all forms of spontaneous evacuation the most unsatisfactory. It usually occurs through or above the fifth interspace, and œdema of the chest wall is a common signal. In a few cases recovery follows, but the majority die of exhaustion or waxy disease. Five cases of this have occurred in my hundred cases. All had been discharging a considerable time before coming to me. In two cases the pus had escaped through the eighth interspace in the mid-axillary line. The other three cases were discharging, one through the second interspace below the centre of the clavicle, the other two through the fifth interspace. Four of these cases were cured by Estlander's operation, the fifth by ordinary incision and drainage. The last case, E. E., æt. 26, had been under treatment for some months prior to consulting me, and had undergone two operations for necrosis of the ribs. On examination, a tortuous sinus existed under the right breast, and this led through the sixth interspace into the pleural cavity. A counter-opening was made in the mid-axillary line in the fifth interspace, and a large-sized drain inserted. The breast was reflected upwards by a semilunar incision, the sinus opened up and scraped, and plugged with iodoform gauze extending up to but not through the opening in the chest wall. A tedious recovery took place, but no deformity of the chest resulted. This successful result I attribute to the valvular nature of the opening through which the pus was discharged, preventing septic infection of the pus which was in the pleural cavity.

5. *Surgical treatment* offers the best results, immediate and remote.

My cases proved that uncomplicated empyema treated with proper surgical care is not a serious disease, for of a hundred cases all but three recovered. The deaths were due to bronchitis, marasmus, and cerebro-spinal meningitis respectively.

If the saving of life is satisfactory, the perfect recovery of these cases is more satisfactory still. I have measured and examined the chests of most of them months after operation, and, except for the scar, it is impossible to say which side has been affected.

Empyema has never to my knowledge been followed by tuberculous lung disease. Recovery has been complete and permanent in all uncomplicated cases. In one of my cases, a boy æt. 9, the right lung was compressed for six months before an operation was allowed by the parents, and a marked lateral curvature was present. Two months after the closure of the wound the right half of the chest measured the same in circumference as the left, and all trace of spinal curvature had disappeared. Such a result can surely only be predicted if the irritation of putrefaction or irrigation be avoided, either causes chronic thickening, induration, and subsequent contraction of the inflamed pleura, with deformity of the chest and spine.

The average duration of treatment from the date of the incision to the completion of healing was thirty-three days. The shortest period was twenty-one days, the longest sixty-one days.

Treatment.—There is no medicinal treatment that will cure empyema. It requires surgical measures as an ordinary abscess does. Occasional recoveries follow *aspiration*, and in my earlier cases I tried it, but very seldom with good result. In a large percentage of cases the pus reaccumulates rapidly, and I soon concluded against further trial of the aspirator.

Aspiration is, however, of service in one class of case—in patients who are suffering from dyspnœa, due to a large collection of pus, or in whom some complication, such as double empyema or severe bronchitis, renders operative interference inadvisable. In such cases it is to be resorted to only to tide the patient over a trying time, and as soon as the urgent symptoms disappear the chest should be opened and drained. In the following case I employed this method with satisfactory result:—

Case 4.—G. C., æt. 10 months. First seen 14th November. A history of "bronchitis" for a fortnight. Left chest full of fluid, which the needle proved to be pus. There was capillary bronchitis on the right side, with great dyspnœa. I aspirated and withdrew a pint of pus from the left chest, with marked improvement to the respiration. The child's condition improved, and on 20th November I opened and drained the left pleura of 1 pint of pus. Convalescence was rapid.

The Operation.—The patient, surgeon, instruments, and assistants are prepared as for an aseptic operation with the most scrupulous care. An anæsthetic in the case of children is always advisable, though in the case of adults with a very full chest, the

operation can be done, and with much greater safety, with the aid of a local anæsthetic.

Chloroform is the best anæsthetic for the purpose, but must be administered with special care, and *during the whole of the operation the patient should lie on the back.* I have seen most serious effects produced by turning the patient over on to the sound side during the operation. This may be avoided by drawing the patient to the edge of the table, so that the side to be incised projects over the edge.

The situation of the incision.—In all cases it is advisable before incising the chest wall to explore with a needle to ascertain the position of the pus. This was very forcibly impressed on me by the following case :—

Case 5.—E. W., æt. 3, had broncho-pneumonia three weeks ago, and as the child's health and the condition of the chest did not improve, its medical attendant explored the left chest with an exploring needle, and found pus in the fifth interspace in the posterior axillary line. Two days after, he asked me to operate. Unfortunately, the exploring needle had been forgotten, and I therefore made the central point of the incision at the spot indicated by the previous needle puncture. On opening the pleural cavity, no pus was found, and though I searched in all directions with a pair of sinus forceps the abscess could not be detected. The incision was therefore closed, and next day, with an exploring needle I found, after many attempts, a small collection of pus (about 2 oz.) in the fifth interspace about 2 in. from the spine. A drainage tube was inserted, and the case did well.

The point I select when possible is in the sixth interspace just in front of the posterior axillary line. A vertical incision is first made through the skin, then a horizontal one through the muscles along the upper border of the seventh rib, into the pleural cavity. A pair of sinus forceps is then introduced into the pleural cavity, by opening and withdrawing which the wound is dilated. Before all the pus has escaped an indiarubber drainage tube, a $\frac{1}{4}$ in. in diameter and 2 in. long, is inserted. The tube is perforated for the inner third of its length only, for if there are any openings in the outer part of the tube it frequently becomes blocked from granulations growing into them. A safety-pin passed through the outer end prevents the drainage tube from slipping in—an accident apparently of not infrequent occurrence. As soon as the discharge has become small in quantity, half the tube may be cut off, the

remainder retained for a few days. For the first days after removing the tube I am always anxious, and if the cough returns, with some rise of temperature or vomiting, I explore the wound with a probe, lest the outer opening should have closed too quickly.

In long-standing empyema, the diaphragm may be wounded in re-introducing the tube if care is not taken. The collapsed lung allows the diaphragm to ascend into the chest, and it lies closely apposed to the chest wall; being softened too, it is easily torn and the abdominal cavity may be opened.

The wound is dressed with corrosive gauze and wood-wool wadding, and the dressing is kept in place by a domette bandage fixed by braces over the shoulders, and prevented from rising to let air pass underneath by three or four turns of elastic webbing bandage. It is in all cases advisable to dress the wound two or three times during the first twenty-four hours, in which the discharge is most profuse. The dressings after this are changed daily till the second week, when every other day is generally sufficient. The guide to frequency of dressing is the amount of discharge. The bandage should never be allowed to be stained longer than can be avoided. During the third week, in a large proportion of cases the tube can be removed and the wound rapidly closes. The discharge should be serous after the third or fourth day. If it is purulent, this indicates either that the tube is not acting efficiently or that the wound has become septic. Irrigation is unnecessary and hurtful. Even the most foetid cases become sweet after a few days' good drainage and careful dressing. As an aid to assisting the lung to expand, immediately the patient is out of bed, he should be encouraged in the use of gymnastic appliances—trapeze, horizontal bar, etc., and in making respiratory efforts such as are required in running upstairs, or in playing wind instruments. In a certain proportion of cases, a long narrow sinus which discharges a small quantity of serum, persists in spite of this treatment. The line the sinus takes is generally backwards and upwards. I have found that by making a counter-opening posteriorly midway between the end of the sinus and the original opening, inserting a drainage tube and allowing the first wound to close, that the closure of the sinus is speedily effected.

In four cases I have performed Estlander's operation, and the notes sufficiently indicate the class of cases in which this operation is called for. In all instances the wound had been allowed to become septic. In all there was persistent purulent discharge

for from two and a half to eight months, and this had reduced the patients to a weak and emaciated condition. The side was contracted, the ribs close together, and the lung appeared bound down.

A semilunar incision with its convexity downwards was made in the lateral chest wall, and the soft parts dissected off the ribs and turned upwards. An incision was next made through the periosteum covering the ribs from the third to the eighth, and a varying amount of bone excised, from 2 to 4 in. A free opening was then made through the pleura, and a counter-opening posteriorly. A drainage tube was passed from one opening to the other, and in shortening it the tube was left in the posterior opening. All the cases did remarkably well, but in all considerable deformity remains. My conclusions are :—

That pleuro-pneumonia is the most frequent cause of empyema.

That empyema rarely follows pleurisy with effusion.

That in adults empyema is more likely than in children to be secondary to serious lung disease.

That the diagnosis of empyema is not always easy, for it may be ushered in by symptoms pointing more to the abdominal than the thoracic cavity, and the physical signs resemble closely those of consolidation.

That the exploring needle should be used in all doubtful cases, and, if needful, in more than one place.

That there is no danger in using the needle with proper care.

That the immediate prognosis of an uncomplicated empyema treated by incision and drainage is good. It is worst when allowed to burst through the chest wall.

That the remote prognosis is excellent. No deformity follows recovery, and no sequelæ are to be anticipated.

That the best prognosis can only ye given in cases where an aseptic condition of the pleural cavity can be assured by antiseptic operation and antiseptic after-treatment.

That the anæsthetic should be carefully administered, and the patient not turned over during its administration.

That in adults it is safer to do the operation without general anæsthesia.

That aspiration is disappointing as a curative agent, but is useful in some cases temporarily.

That incision and drainage with antiseptic precautions may be trusted to cure all cases of curable empyema.

That irrigation is unnecessary and dangerous.

That rib resection is needful in some neglected cases, but in the generality of cases it is meddlesome surgery.

Meeting VI.—March 4, 1908

Dr JAMES RITCHIE, *President, in the Chair*

I. EXHIBITION OF PATIENTS

1. *Mr Alexis Thomson* exhibited a boy, aged 10, who complained of inability to completely extend the forearm at the elbow-joint. There was no evidence of disease and no definite history of injury. A skiagram showed, however, that the upper epiphysis of the ulna had been partially separated and displaced forwards to a very slight degree, thus accounting for the mechanical arrest of the movement of extension when nearing its extreme limit.

2. *Mr George Chiene* exhibited two patients on whom he had operated for PERFORATED GASTRIC ULCER in which the abdominal wall was completely closed at the operation.

The two patients shown to-night were admitted to Mr Cathcart's wards, one 4, the other 2½ months ago, both suffering from ruptured gastric ulcer. I think they may be of interest to the Society as they illustrate certain points regarding the resistant power of the peritoneum against extravasation of the contents of the stomach, which Mr Dowden drew attention to at the January meeting. At that meeting he showed a patient on whom he had operated for perforated gastric ulcer to illustrate the result of *early* complete suture of the abdominal wounds. In the case which he showed it may be remembered that on inspecting the abdominal cavity about forty-eight hours after the initial operation, he found it in such a satisfactory condition that he completely stitched up the abdominal wounds. The cases shown to-night differ in that the abdominal cavity was completely closed at the time of the operation, no drainage whatever being employed. In both cases rupture had occurred about four hours before the operation, in both the ulcer was found on the anterior aspect of the stomach towards the pyloric end and near the lesser curvature. The treatment was exactly similar. After closing the ulcer the extravasated fluid, which was localised, was carefully removed by sponging with gauze until the parts involved were apparently quite dry, the abdominal wound was then sutured

Primary union occurred in both cases without any complications.

These cases not only illustrate the point drawn attention to by Mr Dowden, but also illustrate the great importance of early operation in cases of perforated ulcer.

I am sorry that discussion is not allowed when cases are shown, as some might question the treatment adopted; but the cases speak for themselves, and the possibility of secondary adhesions with consequent interference with the functions of the stomach is not nearly so likely to occur as in those cases in which local drainage is employed; whereas, owing to the early time at which the cases were seen, any attempt to drain the pelvis would probably have disseminated the extravasated contents. It was not thought necessary to perform gastro-enterostomy in either case, as the ulcer was not sufficiently near the pylorus. Both men have kept well since the operation, and both are doing their usual work.

II. Exhibition of Specimens

Dr F. M. Graham exhibited a photograph and skiagrams of a case of ABSENCE OF BOTH FOREARMS.

GENERAL DESCRIPTION

Patient (a native of Hankow) with *scoliosis of spine* and *maldevelopment of arms.*

Age 56.

Height 4 feet 6 inches.

No one else in his family was deformed. His deformities have existed from birth—of chief interest is the *congenital absence* of the bones of the *forearm.*

Right Arm

Shoulder-joint defective. Upper arm cannot be raised from side, but only has antero-posterior movement.

Right humerus 9½ inches long. *At lower end* shows a spine of bone projecting downwards and inwards, 1½ inches below level of external condyle. Triceps is apparently attached to this spine. In front of lower end is a mass of small bones, which slides on the humerus.

In front of these bones are the *metacarpals* and phalanges of two complete fingers, provided with flexor and extensor tendons; lying

to the radial side are the rudimentary bones of a third digit which has no muscles.

Elbow-joint moves 40°.

From front of elbow to finger tip is 7 inches.

Skin of palm comes to within 1 inch of humerus.

Left Arm.

Movements of shoulder defective as in case of right arm.

Left humerus 7 inches long. At lower end a projecting spur of bone passes downwards from the inner side of the bone to 1½ inches below level of external condyle. *Biceps* is distinctly attached to a bone lying in front of lower end of humerus.

Triceps is attached to the projecting spur.

Bones of carpus and two perfect fingers and one rudimentary digit present, the latter having no separate covering of skin. The skin of the palm extends half-way down the metacarpals. From point of elbow to finger tip is 6¼ inches.

III. Original Communications

1 DIVERTICULA OF THE ALIMENTARY TRACT WITH PARTICULAR REFERENCE TO THOSE MET WITH IN THE ILEUM RESULTING FROM AN ACCESSORY PANCREAS AND FROM TUBERCULOSIS

By Allan Thomson, F.R.C.S.Ed., Assistant-Surgeon, Edinburgh Royal Infirmary

Diverticula or pouchings are met with in relation to so many of the tubular and saccular structures of the body, that their consideration would lead me far beyond the limits of the present occasion were I not to restrict myself to those connected with the alimentary tract. Diverticula are met with almost throughout its whole length, but occur with varying frequency in the different parts of the digestive tube. They are, for example, comparatively common in the pharynx, gullet, lower part of the ileum, and in the sigmoid colon; they are comparatively rare in the stomach, duodenum, jejunum, vermiform appendix, and in the rectum; so far as I am aware, their occurrence in the cæcum or ascending colon has not been noted. It was formerly the custom to speak

of true diverticula, which consists of a pouching of all the coats of the digestive tube, and of false diverticula, in which the mucosa and serosa are pouched through a gap or defect in the muscularis. The demonstration of acquired pouches in which all the coats are represented has made this distinction invalid, and we now speak of *congenital diverticula*, in which the protrusion depends on the persistence of fœtal structures such as the visceral arches or the Vitelline duct, and of *acquired diverticula*, in which the chief etiological factor is any condition that impairs the integrity of the muscular coat of the digestive tube.

Diverticula of the pharynx and œsophagus are comparatively common. Those springing from the pharyngeal wall take origin from the inner portion of the second visceral cleft and open in the immediate vicinity of the tonsil. In the unique specimen described by Morrison Watson, to which my attention was directed by Professor Cunningham, the inner opening was situated behind the tonsil, while the lower blind expanded extremity reached down to the episternal notch; its wall did not contain any circular muscular fibres, but possessed a single layer of striped fibres arranged parallel to the long axis of the tube.

A much more common seat is *at the junction of the pharynx with the œsophagus*, where there is an anatomical weakness due to the divergence of the longitudinal muscular fibres, which run forwards as they ascend in the shape of two lateral bands to become attached to the cricoid cartilage. The weakness is most marked in the middle line posteriorly, where the majority of the so-called "*pressure pouches*" occur, but the weakness also effects, although to a less extent, the lateral walls of the gullet in this situation. There is a characteristic example of a pouch at this level springing from the left lateral wall of the tube, in the St. Thomas's Hospital Museum, London. Butlin, who was one of the first in this country to write on these pouches, referred to the absence of muscle fibres in the wall of the pouch observed by him, and to the extraordinary elasticity of the pouch, so that it could be easily distended to two or three times its natural size.

Diverticula of the gullet resulting from defects in the muscular coat are met with at *other parts of the tube*, and of this I am able to show an instructive example. It is a pouch about the size and shape of an ordinary thimble, projecting from the right antero-lateral aspect, at the junction of the middle and lower thirds of the thoracic œsophagus. The wall of the pouch is

entirely deficient in muscular fibres, and so thin as to be translucent. The entire gullet when laid open and held up to the light shows numerous irregular areas in which the muscular coat is abnormally thin. This specimen was obtained from the body of a man, æt. 70, a confirmed drinker, who succumbed to pulmonary emphysema and cirrhotic kidneys.

Another site of œsophageal diverticula is in the *region of the bifurcation of the trachea* and crossing of the left bronchus: these have been described as "*traction diverticula*," because they were supposed to result from the contraction of cicatricial tissue formed in relation to disease of the adjacent lymphatic glands. They are believed, however, by Ribbert [1] to be the result of a congenital defect in the muscular coat arising at the time of separation of the air passage from the gullet.

Pouches which form in the process of encystment of a *foreign body* which has been impacted in the œsophagus may form at any part of the tube, as they are known to form, from a similar cause, in other parts of the alimentary tract as a result of the weakening of the muscular tissue by inflammatory changes.

In the stomach, diverticula are so rare as to be of little or no clinical importance. A few specimens are recorded, in which the stomach has presented one or more congenital pouchings which have been compared to the compartments of the stomach met with in ruminants. The only other instances recorded are those of minute diverticula in the vicinity of the pylorus associated with the presence of an accessory pancreas.

In the duodenum, diverticula are also rare, and would appear to have only an anatomical interest. My attention was directed to them by Professor Cunningham. Two varieties are met with. The first resembles those which occur in the stomach in relation to an accessory pancreas. In the second, a pouch about the size of a pigeon's egg or a walnut springs from the median side of the gut a little above the ampulla of Vater. In the cases described by Rolleston and by Targett, the sac, which was embedded in the substance of the pancreas, was devoid of muscle fibres. In view of their situation in the vicinity of the diverticula which are concerned in the formation of the liver and pancreas it is believed they owe their origin to the persistence of supernumerary pouches formed early in foetal life. C. M. Jackson describes a duodenal pouch found in the body of a man, æt. 50, springing

[1] *Virchow's Arch.*, 1898, Bd. cxxvii.

from the upper edge of the transverse portion of the duodenum, and possessed of a rudimentary muscular coat, which would appear to have had a similar origin to those described by Rolleston and Targett.

In the jejunum. Although the jejunum is not infrequently the seat of an accessory pancreas, I have not seen the record of one which was associated with a diverticulum. Multiple small acquired diverticula are stated to have been met with by Hansemann,[1] as many as four hundred being counted in the jejunum, ileum, and sigmoid.

Before considering those met with in the lower part of the ileum which form the main subject of this paper, I shall briefly refer to those met with in the remaining portions of the alimentary tract.

Diverticula are not infrequently found *in the appendix vermiformis* when operating for recurrent appendicitis. They were made the subject of an interesting article by v. Brunn some three years ago. I have met with two examples in 130 cases. The muscular coat is destroyed or replaced by granulation or scar tissue at one point, and the mucous membrane, with what remains of the other coats, is herniated through and forms either a small pouch or a cylindrical diverticulum lying parallel to the appendix and forming with it a structure resembling a double-barrelled gun. Clinically they are associated with recurrence of attacks at frequent intervals, and the operation for the removal of the appendix is usually a difficult one on account of the extent of the adhesions.

Acquired diverticula are commonly met with *in the descending colon and sigmoid.* They are usually multiple, of a minute tubular character, and apparently result from the protrusion of the mucous membrane through defects in the circular muscular coat. The most complete description of these I have been able to find is by Dr. L. B. Wilson in a paper by William J. Mayo.[2] All the diverticula are described as being small, with atrophied mucosa, thickened submucosa, and no muscular coat. Examination of the circular muscular coat of the sigmoid in the vicinity of the diverticula showed areas of irregular thinning, in which the muscular fibres seemed to be entirely absent. Wilson points out that their hernial nature was further shown by the fact that

[1] Quoted by Nothnagel. Reference not given.

[2] "Acquired Diverticulitis of the Large Intestine."

all had penetrated the haustra of the sigmoid well away from the tenie of the longitudinal muscularis. It would appear to be uncertain whether the deficiency of the circular muscular fibres is congenital or the result of atrophy from the pressure of hardened faces.

Diverticula of the sigmoid and descending colon undergo pathological changes more frequently than those met with in other parts of the intestinal tract, because of the difference in the character of the bowel contents. The most important clinical conditions associated with these diverticula are inflammatory troubles in the left iliac region, often closely simulating appendicitis, and, like this, terminating in localised abscess or in diffuse forms of peritonitis. In another group of cases there results an inflammatory thickening of the sigmoid associated with narrowing of the gut and the recognition of a tumour in the left iliac fossa which is liable to be mistaken for malignant disease. In some cases the formation of an abscess has resulted in the production of fistulous communications with the bladder.

True diverticula of *the rectum* are extremely rare: only a few instances are recorded, and with such meagreness of detail that one is not warranted in drawing conclusions from them, except that they appear to owe their origin to errors in development.

Diverticula of the ileum.—In studying diverticula of the lower ileum, it is necessary at the outset to make an attempt to differentiate the congenital form which is associated with the name of Meckel from the acquired varieties. Meckel's diverticulum when of the "finger-of-a-glove" type and lying free in the peritoneal cavity, can usually be distinguished from the acquired forms by its frequently possessing a mesentery, by its containing all the coats represented in the normal ileum, and by the absence of any lesion at its free extremity capable of giving rise to a pouching of the bowel such as is so constantly found in acquired diverticula. If a Meckel's diverticulum is the seat of disease, the latter is secondary and not the cause of the protrusion. That it always springs from the bowel opposite the attachment of the mesentery cannot be relied upon in the differentiation from acquired diverticula, for there can be no doubt that the latter may present a similar relationship, and there is as little doubt that an acquired diverticulum may be solitary.

The occurrence of acquired diverticula of the "finger-of-a-glove" type has only recently been admitted. A cursory glance

at the literature reveals the fact that these have been frequently mistaken for the Meckel variety, and have been recorded as such: the error is excusable, for the resemblance is remarkable. The observation in the ileum of two diverticula, only one of which could be a "Meckel," was an important factor in its correction, and various stages in the formation of the acquired diverticle were then observed. It has been my good fortune to meet with two examples which, to my mind, go far to confirm the accuracy of the view that many diverticula of the "finger-of-a-glove" type are of the acquired variety.

Case 1.—*Acquired diverticulum of lower ileum due to a localised tuberculosis of the wall of the bowel.*—This specimen was removed in the course of a laparotomy from a woman, æt. 65 (seen in consultation with Dr Hope Fowler), who had had threatenings of intestinal obstruction, with recurrent colicky pains referred to the lower part of the abdomen on the right side. On opening the abdomen, some attenuated adhesions were found between the cæcum and the anterior abdominal wall, and between the different coils of the lower ileum. The cæcum, appendix, and the glands in the adjacent mesentery were normal. On pulling out the lower ileum, several coils were encountered, the loops of which were approximated by attenuated adhesions, and about 2 ft. from the lower end of the ileum a diverticulum was found about the size of the little finger and about 1 in. in length, springing from the bowel midway between the mesenteric attachement and the border opposite to this; in consistence it felt remarkably solid, as if there was a growth in its walls. It was removed in the ordinary way with the appendix crusher, and the gap closed by a purse-string suture. She made an uneventful recovery, and is now, after an interval of five months, in good health.

Sections of the diverticulum were made, and showed the following appearances: Towards the free extremity, there are a number of rounded foci of giant-celled tuberculosis scattered irregularly through the different coats of the bowel. Those in the deeper layers of the mucous coat and in the submucous coat appear to be the oldest. There are several in the muscular coat, and at one point these had completely destroyed and replaced the circular muscularis. There are a number in the subperitoneal tissue, these having evidently disseminated by way of the lymphatics. The tuberculous foci show a large number of typical giant cells and a considerable degree of central necrosis, while in their vicinity the vascularity is greatly increased, and there is little fibrous tissue, so that one feels justified in concluding that the tuberculous lesion,

although of the chronic variety was not of very long standing and that it was progressive. It would also appear that the tuberculous infection had extended from the lumen of the intestine outwards and that the spread of the infection to the peritoneal coat was responsible for the formation of the adhesions observed in the vicinity of the cæcum and cæcal segment of the ileum. It seems reasonable to ascribe the threatenings of obstruction and colicky pains to the presence of the diverticulum and to the adhesions in its neighbourhood, these exciting irregular and exaggerated peristaltic movements.

It is undeniable that it is very rare to have tuberculosis limited to so small a segment of the ileum alone, but it is my opinion that the tuberculous lesion was the primary one, and that by its destructive effects on the circular muscular coat it had caused the pouching of the bowel described.

Case 2. *Acquired diverticulum of lower ileum with necrosis of pancreas, causing symptoms mistaken for those of acute appendicitis.* The patient from whom this specimen was removed was a stout man, æt. 33, a grocer, seen in consultation with Dr Dickie on 2nd March 1905. The history showed that two years before he had had an attack attended with abdominal pain, shivering, and vomiting, regarded by his doctor as appendicitis. The present attack began suddenly on the afternoon of the 2nd March 1905, with shivering, inability for food, pain referred to the umbilicus, and vomiting; after much straining he passed a small motion mixed with blood. These symptoms passed off after being put to bed, but on the evening of the following day the pain returned, the temperature rose to 103°, there were pain and tenderness in the right iliac fossa, and the bowels moved twice. The loading of the abdominal wall with fat rendered the examination difficult, but there was distinct tenderness and impairment of respiratory movement in the right lower quadrant to lead to our making a provisional diagnosis of peritonitis, probably originating from the appendix, and he was removed to a nursing home for operation.

On opening the peritoneal cavity thirty-four hours after the first onset of symptoms a small amount of clear serum escaped. The appendix did not show signs of recent inflammation, but as it was bound down about its middle and likely to give trouble in the future, it was removed. The lower coils of ileum were then pulled out at the wound, and, about 18 in. from the ileo-cæcal valve, a diverticulum was found of the "finger of a glove" type, presenting a bulbous enlargement, resembling in shape the glans penis. Its peritoneal coat showed a thin deposit of fibrin but no undue vascularity; it was removed by the same method as is employed for the

Complete Longitudinal Section of Diverticulum with Accessory Pancreas. × 5.

a, Extreme apex of diverticulum; *b*, Fat: *c*, Larger portion of accessory pancreas embedded in extraperitoneal fat; *d*, Smaller portion of accessory pancreas; *e*, Blood-vessels; *f*, Longitudinal muscle fibres of coat of bowel; *g*, Circular muscle fibres; *h*, Mucosa with villi and solitary glands; *i*, Lumen of diverticulum.

PLATE I.—TO ILLUSTRATE MR ALEXIS THOMSON'S ARTICLE.

removal of the appendix. Further exploration of the abdomen yielded negative results, and the wound was closed without drainage. He made an uneventful recovery. Three years have now elapsed since the operation, and he has had no return of the abdominal symptoms. His health has been excellent, and although thinner than formerly, he has been able to undertake without difficulty the management of an exacting business.

The diverticulum in its contracted state (due to fixation in alcohol) measured 3 cms. in length; the tubular portion measured 1 cm. in diameter, while the club-shaped extremity measured 1.3 cm. The tubular portion presented the appearances of normal small intestine; the club-shaped portion appeared to be composed of fat; there was a shallow constriction intervening between the two portions.

On section, the club-shaped portion was seen to be soild and to constitute fully one-half of the entire diverticulum; its centre presented a lobulated appearance as if composed of gland tissue, while the cortex consisted of fat.

The relations of the diverticulum to the tumour at its apex are well seen in Plate I. At the distal end of the tubular portion, and confined within the longitudinal muscular coat, is a minute, well-defined mass of pancreatic tissue, the lobules of which are arranged as in the normal pancreas, and are provided with an average number of ducts. One of the peripherally placed lobules, however, shows a considerable increase in the amount of stroma and an increase in the number of the smaller ducts and of the medium-sized blood vessels. At the blind end of the tubular portion of the diverticulum, the circular muscular coat of the bowel is absent.

The larger mass of pancreatic tissue lies outside the muscular coat, and is embedded in extra-peritoneal fat; the latter, in its turn, is invested with a thin membrane derived from the serous covering of the bowel, and is provided with an excessive number of large, thin-walled blood vessels. The main mass of pancreatic tissue sends out irregular prolongations of its substance into the fat, and one of the prolongations extends so far as to become connected with the investing serous membrane. In the centre of the glandular mass is the section of a large duct, lined with columnar epithelium, and containing shed epithelium, nuclei of leucocytes, and threads and clumps of hyalin material. The epithelial lining of the duct is much subdivided, and appears to be

engaged in growth like that observed in tubular adenomata. The size of this duct and of its branches is excessive in proportion to the amount of gland tissue surrounding it. The lobules of gland tissue surrounding this centrally placed duct differ greatly in structure and arrangement from those of the normal gland. In many of the lobules there is an excessive amount of small ducts; in some of them this has extended to the almost complete replacement of the pancreatic acini, and gives the affected lobule the appearance of a tubular adenoma. This replacement of pancreatic parenchyma by newly formed ducts is widely distributed. Similarly widespread is the replacement of the pancreatic acini by solid masses of epithelial cells arranged in round or oval alveoli, presenting an arrangement of tissue elements resembling that of an alveolar cancer. Transition forms are also met with between the most minute of the newly formed ducts and the solid masses of the cells which give the cancerous appearance to the parts thus affected. It is not possible to identify any islands of Langerhans, although it must be confessed that the appearance of islands is closely simulated by those alveoli which are filled up with what we are inclined to regard as cancer cells. The resemblance to islands is increased by the existence of capillary blood vessels and of a delicate stroma between the epithelial cells.

The extent of replacement of pancreatic acini varies in the different lobules. In some they are fairly well preserved; in the majority they have to a large extent disappeared. Under higher magnification the process of destruction and replacement of the normal gland acini by newly formed ducts and by alveoli filled with epithelial cells can be distinctly traced (Plate II.).

The stroma shows a great departure from the arrangement and structure of the normal pancreas. It is greatly increased in amount, so that the lobules and acini are widely separated from one another, and it bears an excessive number of large blood vessels possessing walls of considerable thickness. In certain parts, especially around the proliferated ducts, the stroma is largely composed of an interlacing meshwork of smooth muscle fibres, cut in longitudinal and transverse sections. In other parts, and especially where the replacement of pancreatic acini is in active progress, it is richly permeated with small cells, these being here and there so abundant as to impart to the stroma the characters of granulation tissue; the latter is rich in capillary

Portion of accessory pancreas. × 150. Showing isolated remains of pancreatic acini; alveoli filled with epithelial cells. ? Cancer.

Portion of accessory pancreas. × 150. Showing proliferative changes in ducts, replacement of pancreatic acini, alveolar spaces filled with epithelial cells, increase in stroma.

PLATE II. – TO ILLUSTRATE MR ALEXIS THOMSON'S ARTICLE.

blood vessels, and may be observed to be replacing by a process of eating up the pancreatic gland tissue.

Near the junction of the tubular portion of the diverticulum with the solid bulbous extremity, and situated on the deepest part of the submucous coat, there are a few scattered masses of epithelial cells, which suggest a cancerous infection of the lymphatics.

Here, then, we have an accessory pancreas consisting of two portions—one, much the smaller of the two, included within the longitudinal muscular coat of the bowel, and retaining for the most part the characters of the normal pancreas; the other, much the larger, is embedded in subperitoneal fat, and presents changes of a complicated character. The most prominent of these changes are: An active proliferation of the duct epithelium, resulting in the formation of a large number of new tubes; a change in type of this proliferation, whereby the newly formed epithelial cells are collected into solid groups, and occupy spaces which come to resemble those of an alveolar cancer; an extensive replacement of the pancreatic glandular tissue by newly formed tubes, by alveoli filled with epithelial cells, and by a small-celled infiltration of the stroma; and, finally, a great increase in the amount of stroma and in the number of blood vessels, the stroma consisting in some lobules of interlacing bundles of smooth muscle fibres, in others of connective-tissue fibres, and in others again of a small-celled infiltration resembling granulation tissue.

It is difficult to focus all the inferences to be drawn from a study of the observations above recorded; but I shall attempt to discuss these under the headings of the clinical features, the nature of the diverticulum, and the interpretation of the changes in the accessory pancreas.

The clinical features.—Although the operation was undertaken on the understanding that the peritonitis proceeded from the appendix, a survey of the clinical features (*vide ante*) shows that they departed in some respects from those of appendicitis, while they resembled the features observed in cases of acute pancreatitis, not only in the attack which led to the operative interference, but in the milder attack which occurred two years before. Is it not possible that the changes which were taking place in the accessory pancreas led to alterations in the character of its secretion, and that this, unable to discharge into the intestine, escaped into the peritoneal cavity and caused the phenomena of peritonitis? I am unable to explain the clinical features and the immunity from

symptoms subsequent to the operation on any other hypothesis.

The nature of the diverticulum.—The fact that it was of the "finger-of-a-glove" type, and sprang from the ileum about 18 in. above the ileo-cæcal valve, would suggest that it owed its origin to a persistence of the remains of the Vitelline duct. I have no doubt, however, that it is an acquired diverticulum, resulting from the presence of the accessory pancreas. This structure consisted of two portions, one of which lay within the longitudinal muscle, and replaced the circular muscle at a point which ultimately became the apex of the protrusion, and it is by the yielding of the unsupported longitudinal muscle fibres to the pressure within the intestine that the protrusion was brought about.

The changes in the accessory pancreas.—It has been seen that these are of a complex nature. Certain of them, such as proliferation of the duct epithelium and multiplication of the ducts, increase in the amount of stroma with the appearance of interlacing bundles of non-striped muscle, are commonly observed in the accessory pancreas, wherever situated, and has led to their being described as cysts, adenomata, cyst-adenomata, adeno-myomata, and myomata in the wall of the stomach or intestine. I shall throw on the screen an accessory pancreas in the subcutaneous coat of the stomach from a man æt. 73, observed by my assistant, Dr Graham, in which, side by side with normal pancreatic gland tissue, there are numbers of newly formed ducts closely resembling the arrangement of parts seen in tubular adenomata of the breast. I have also demonstrated in my own specimen the new formation in the stroma of smooth muscle similar to that in the muscular coat of the intestine, and either derived from this or from the muscular coat of the ducts. Thorel, who has investigated the origin of the muscular tissue in the stroma of the accessory pancreas, believes that they may arise from three different sources, namely (1) The muscular fibres accompanying the larger ducts; (2) those in the walls of the blood vessels; and (3) from the fibres of the muscularis mucosæ.

A striking feature in my specimen of accessory pancreas is the evolution from the proliferated epithelium of the smaller ducts of what I am inclined to regard as an alveolar cancer, and this, so far as I am aware, has not been described before. It appears to me to be an observation of considerable interest, as shedding light on the anatomical beginnings of cancer, and as affording a remarkable illustration of Cohnheim's doctrine of the origin of tumour growths from embryonic residues.

ILLUSTRATIONS

The plates are reproduced from photographs made by Mr Richard Muir, assistant in the Department of Pathology, Edinburgh University

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2. THE PROVISION OF SANATORIA FOR THE CURATIVE TREATMENT OF PHTHISIS AMONG THE POORER CLASSES.

By Alexander Walker, M.D., Edinburgh

The curative treatment of phthisis, even if confined to one class of the community and to one method—the sanatorium—is too big a subject to be discussed in a few pages. I propose, therefore, to leave out of view aspects of the question which are no less important than those which I include, because I feel myself less competent to deal with them than those who have practical experience to guide them. Among these are the difficulty of selecting suitable cases, for which the dispensary system,

honourably associated with the name of Dr R. W. Phillip, is the most successful method—the indoor treatment of the patients, regarding which he and Dr Caverhill are acknowledged experts; and the unsolved but extremely important question of the after-care of discharged cases. All I shall say on these points is to protest against them being used as excuses for doing nothing, and to express my belief that the first and last will be solved by time and education, whilst the second is now rapidly approaching scientific precision and complete success.

For time and other reasons I will confine myself to an attempt to show that, on a priori grounds, the sanatorium is the most promising method of treating early cases of phthisis—that it must be placed within the reach of the poorer classes among which the disease has its stronghold and from which it spreads, and that, to enlist the public sympathy and support, it must be erected and run on inexpensive lines consistent with efficiency, and depend on the free-will offerings of all classes of the community, and not on a compulsory levy made by the State. I leave untouched the thorny question of notification, and only incidentally refer to the disputed points regarding human and bovine tuberculosis.

First, let us consider the reasons why sanatoria may be looked upon as the most promising means of treating phthisis in its early stage.

You will all agree that the tubercle bacillus discovered by Koch in 1882 is the true and only cause of phthisis, and that its discovery has revolutionised our opinions regarding the causes and treatment of the disease. The life-history of the parasite is now well known. Its chief characteristics are these. It is killed by direct sunlight quickly, and by diffused light more slowly. It does not grow except under artificial conditions, and remains alive with difficulty outside of the animal body, but when dried it possesses a vitality which has affinities to the spore-bearing bacteria, and can withstand extremes of heat or cold as well as trituration. It finds the healthy body, especially when its owner is well fed and clothed, and living in a healthy environment, almost impregnable to attack. It is confined practically to one mode of dissemination, namely, the infected sputum of advanced cases among men, and to a limited extent to infected milk or flesh from the lower animals. In the moist condition it is easily destroyed by heat and germicides. The avenue by which it finds entrance to the human body has been much disputed. In this

country the natural and direct channel by the inhalation of dust from dried sputum, for the formation of which there is every facility, is generally regarded as the most frequent, though we admit the danger of inhaling floating particles of saliva from the mouth of the patient in the act of coughing, etc. On the Continent opinion is more divided. Flügge and Calmette favour the theory that the dust or "droplets" are swallowed rather than inhaled, and reach the lung through the alimentary system. Von Behring regards the disease as originating from infected milk in infancy, the bacillus remaining latent during early life until puberty or disease wakes it into life. The interim report of the Royal Commission on Tuberculosis seems to suggest that there are two types of bacillus, a human and a bovine, differing rather in virulence than in kind, and interchangeable. They seem to be able to affect the same individual as a "mixed infection," or become modified in transmission, by which a change is brought about in their virulence. It is thought that the human type is increased in virulence in the tissues of cattle, and the bovine decreased in the human tissues. Whatever be the theory, the fact of living and virulent bacilli being found in dust is undisputed, and, whether it is swallowed or inhaled, that they infect the body.

The reasonable inference to be drawn from the above facts and theories is that we have to deal with a foe of subtle and malignant power, but we have on our side as an ally and friend a still greater force—that of Nature. We may not agree with Dr Ransome, that man is not naturally susceptible to the disease, but needs some local injury or debilitating disease to render him subject to invasion; but there is evidence that we are protected by invisible but powerful forces which are directed by Nature. Both outside the body in the benefits of a healthy environment, and inside in the phagocytic cells and the soluble antitoxins of the serum, we are marvellously protected. Proof of this beneficent rôle played by Nature is to be found in the following facts:—

1. That pathology has demonstrated that a large proportion (variously estimated at from 40 per cent. to 90 per cent.) of autopsies on persons of adult life dying of other diseases, shows evidence of incipient phthisis, or other tuberculous disease, which has not infected the body generally.

2. That there has been a notable decline in the death-rate from phthisis, and less so from other tuberculous diseases, during the last fifty or sixty years. This decline has affected the better

classes more than the poorer, and the type of the disease most likely to be influenced by improved sanitation, and better conditions of life, namely, pulmonary tuberculosis. Other tuberculous diseases are more dependent on legislative control of food supplies, and do not show the same decline. The reduction in the death-rate has not been related, until very recently, to any organised attempt to cure or prevent the disease, but is due entirely to the operation of natural laws of health which have been allowed free play.

3. That whilst the bacillus is known to be ubiquitous, especially in cities, it selects its victims chiefly among those with hereditary predisposition, and prefers localities where unhealthy conditions exist.

In view of the above facts, it is safe to conclude that many persons possess either a natural or acquired immunity from phthisis; that a still larger number have a power of resistance which enables them to overcome the disease even after it has gained a footing, and that this power has been greatly increased recently by improved sanitation, better food, etc., which have aided Nature in her beneficent efforts. It is noteworthy that no other infectious disease has responded in the same degree to the operation of natural laws, but have either required costly measures of prevention, or still resist all efforts to suppress them.

The question has been gravely argued whether we cannot therefore leave the extinction of phthisis to these natural agencies. Those who honestly (and there are very few) believe that this is possible, seriously underestimate the forces arrayed against such a consummation.

Vested interests on the one hand, and ignorance on the other are obstacles which will bar the advent of a golden age such as More and Richardson dreamt of, for many a generation. Some of the contributing causes of phthisis are physically or morally irremovable, and others would need such drastic legislation that many a Government will come and go before one will be found strong enough to pass it into law.

Meanwhile must we await that far-off event? Let me mention a few objections to delay:—

1. That phthisis is a disease of the poor. There are no statistics available to show the class distribution of disease—a fact rather to be deplored. To satisfy myself that the poor were the greatest sufferers from phthisis, I analysed the death register for two years

(1903-4) in the city of Aberdeen, and found that 90 per cent. of the cases occurred in the poorer class of property. Dr Cullen found that something like this prevailed in Edinburgh. The significance of this fact lies in the facility by which infectious disease travels upwards in the social scale. On the low ground of self-protection we cannot afford delay.

2. That the poorer classes have a strong claim on our philanthropy. They have neither the means nor the knowledge to protect themselves. By the "poorer classes" I am not referring to the "legal poor," which have a claim on State aid already, but that large class which is dragged down from independence and even affluence by this long-protracted disease. An early case suitable for sanatorium treatment is hardly ever met with by the Poor Law inspector except among young children. The honest working man or woman labours on till all hope is past, in the vain endeavour to avoid public charity.

3. That the disease is a most costly one. It is calculated that a tenth part of the national pauperism is due to phthisis, and no disease strains the funds of friendly societies so heavily. It attacks its victims at the most useful and productive period of life, namely, between 15 and 50 as a rule. One in every three deaths from all causes between 15 and 45 years of age is due to phthisis. The results of the compulsory insurance system in Germany are instructive. The banks, which are financially responsible, can compulsorily treat at their own expence any disease which they believe will pay them to cure. On a purely business footing they established about fifty sanatoria, in which, during nine years (1898-1907) they treated over 80,000 working men, and restored over 30 per cent. to health and usefulness permanently. This figure is the net result after four years' continued labour since treatment. The immediate results showed 66 per cent. of temporary recoveries, but half of these relapsed.

4. We have the means to give these helpless folks a chance at a sacrifice infinitely less than we willingly make for less worthy objects. It is said that the means to ill deeds make ill deeds done. Would that the converse were true. From the foregoing considerations I think we are justified in saying that delay is unworthy of us as a wealthy nation.

I am not going to labour the question as to the value of sanatoria. The institution has survived the stage of ridicule and persecution. It is but faint praise to say, as the L.G.B. circular

does, that "this treatment has been more successful than any hitherto attempted." Sanatoria have proved a success among the rich, for whom they are not a necessity, and their success among the poor, to whom they *are* a necessity, may be confidently anticipated. No doubt difficulties have to be faced in securing early cases, and providing for after care, but there can be little doubt that education and experience will in course of time diminish if not remove them.

The great hindrance in the way — and I wish to emphasise this point — is the question of ways and means. Unfortunately sanatoria were founded in the first place for the well-to-do class, and were constructed and maintained on expensive and even luxurious lines. Those which were afterwards instituted for people of small means, or for the poor without payment followed too closely the example set them by the first named. The standard of expenditure both for the erection and maintenance of such institutions for the poorer classes has therefore been far too high, and discouraged the public in supporting them, or in helping to increase their number.

Permit me to give you very briefly the history of a recent effort to establish a sanatorium for the poorer classes.

In the city of Aberdeen there is no adequate provision for the treatment of early cases of phthisis among the poor. In the year 1904 I brought forward a motion in the Parish Council of which I was a member, to invite a conference with the Town and County Councils to inaugurate a municipal sanatorium. The motion was carried, and the invitation accepted by the other Councils. Delegates were appointed from each body, and a date fixed for a Conference in the Town Council Chambers. Meanwhile, with the willing assistance of Professor Hay, the medical officer of health for the city, and Dr Watt, the medical officer of health for the county, I drafted a schedule of twenty-three questions, and sent them to all the then known sanatoria open to the poorer classes. I received a mass of information, which I collated and, after submitting it to the gentlemen above named, published it in the public press, and, in the form of a reprint, circulated it among the members of the three Councils. Not content with documentary evidence, I personally visited almost all the Scotch sanatoria. Meanwhile the subject was brought before the Medico-Chirurgical Society of Aberdeen by Dr Arthur Lister, and there was a general feeling, far more favourable than we expected, ex-

pressed by the members in favour of the scheme he suggested which was on quite as aggressive lines, though not identical with those I was advocating. Dr Lister received from one of his patients an offer of £1000 if a sanatorium was begun within six months, and a sum of money was expected from one of the charitable trusts. There was a considerable amount of public interest created, and the usual flood of correspondence appeared in the papers. The opposition turned almost exclusively on the question of finance. There was no objection to a sanatorium if the money did not come out of the pockets of the ratepayers. Of course the difficulties I have mentioned above were urged and exaggerated by some, who were perhaps ashamed to put their hostility on such selfish grounds, and some considered that Aberdeen should wait to see what other cities were going to do before taking action itself. The hardest argument to meet was the expensive style of the present institutions. Figures were given of the cost of erection and maintenance of certain sanatoria which shall be nameless, and it was argued quite reasonably that Aberdeen could not afford to equip and support such institutions.

I was in hopes of being able to bring forward evidence at the Conference to show that efficient sanatorium treatment could be provided at very much smaller expense than the objectors made out, but I had no opportunity of doing so. About a week before our Conference was to have met, the Local Government Board for Scotland issued a circular, the terms of which made it impossible to go on with the scheme we had in view. Thus, at a stroke, all the labour of months and not a little expense was thrown away, unless we consider the discussion of the subject—from an educational point of view—a sufficient recompense.

Disappointed as I was with this result, it was with mixed feelings that I read that circular. I rejoiced to see that it gave effect, in authoritative fashion, to the opinion of the bulk of the medical profession, that tuberculous disease of the lung was infectious. This is a step forward in the campaign against the disease, and in this matter it is in advance of the English Board. Many other features of the circular commended themselves to me as statesman-like and to the point. But I fear that the attempt to make the provisions of the Public Health (Scotland) Act apply to this disease is to say the least misleading. No doubt the circular is not compulsory, and the Local Authorities need only preserve a masterly inactivity in face of an impossible task. If

the terms of the document had been compulsory it would have been met, in my opinion, with polite but firm resistance as impracticable or premature. There is no attempt to discriminate between the infectious and non-infectious stages which distinguish phthisis from all other communicable diseases, and which makes it unamenable to the same mode of treatment administratively. It is true that the non-infectious stage may be short, as in acute tuberculosis and in a few otherwise normal cases, but this is exceptional. Indeed, in many intermediate cases, and even a few advanced ones, the sputum may be absent, or free from the bacillus, and, even when present, it may be destroyed so as to render the patient perfectly harmless as a source of infection. How far the law would permit the custodians of the public health to go in restricting the liberty of a person who was not actually, but only potentially, a source of danger to the community, may be a nice point for the lawyers, but I for one would not submit to such interference, and I am certain that would be the position of the public generally. Yet the whole of the 1897 Act applies to infectious diseases, and therefore is inapplicable to non-infectious cases of phthisis.

Yet the circular goes on to point out, among the provisions of the Act which it is the duty of the Local Authority to apply, the establishment of a number of institutions which have for their main or sole object the curative treatment of the early and non-infectious cases of phthisis. In no other disease is there an incipient stage prolonged into months or even years, and the Act does not contemplate such a type of disease. It is wholly unsuited to the character of phthisis except in the advanced stage, and even then not satisfactorily. It is suggested that existing sanatoria might be utilised, but it is not indicated what the relations of the Local Authority are to be towards the proprietors or promoters of these institutions. A sub-committee of the Local Authority is to be appointed "as the administrative centre of the whole organisation dealing with phthisis," and to be called the "phthisis committee." If this has to have any such general executive powers, surely sanatoria will come under its control. Unless the distinction between infectious and non-infectious stages of phthisis is recognised and separately dealt with, all institutions receiving patients suffering from it should come under the control of the Public Health Authority. This would include sanatoria, dispensaries, day and night shelters, and hospital wards.

Are the Local Authorities prepared to undertake such a responsibility? Most assuredly not, and it is doubtful whether they should do so even if they could. It is no doubt the dream of the Socialist that all diseases should be a first charge on the national funds. But this idea, like most others coming from the same quarter, would be disastrous to the true interests of the nation. It has been the glory of the last century that it has developed on national lines a grand spirit of charity, expressed in personal service and voluntary gifts in the relief of the sick and helpless poor, and no compulsory and vicarious mode of administration would keep it alive. The tax-collector can never be the almoner of true benevolence, and officialism would extinguish philanthropy. The Local Authority should confine itself to its proper function of the protection of the public health, and leave the curative treatment to voluntary agencies. Still, as curable cases include a certain proportion of infectious persons, there is every reason for the rating bodies subsidising curative institutions, as part of a preventive policy The admitted failure of poor-law administration is an illustration of the inadequacy of State methods in charity work, and the demand for personal and direct helpfulness, such as has been developed at Elberfeld, is evidence of the dissatisfaction even in this country with official methods.

Dr Byrom Bramwell, in his paper last year pointed out the desirability of an amending Act making special provision for the control of tuberculous diseases when in an infectious stage, and I would strongly support this suggestion. It should not be impossible to alter the clauses of the Act of 1897 to carry out this idea, but a fresh measure applicable to this disease would be well worth the labour of drafting, and would immensely simplify the administrative control.

The extravagance which distinguishes public boards, and which is becoming more pronounced since the Socialistic element is increasing in their membership, would lead to an expenditure on sanatoria and other institutions which no country could bear. It would lead also to a further decline in the independence of the poorer classes, now sadly wanting, and encourage the demand for greater privileges at the expense of the State. An illustration of the extravagance of public boards in this direction is to be found in the erection recently by the Liverpool Guardians of a sanatorium at Heswell for twenty-four beds, at a cost of £12,000, or £500 a

bed. The Birmingham Municipality has just bought a site for a proposed sanatorium for £17,000 and in doing so have hung a millstone round its neck. However simple and inexpensive the building may be, this initial expenditure must handicap the institution permanently.

Although I have thus frankly animadverted on the form in which this circular has been issued, I gladly express my admiration for the spirit which has prompted it. The able medical member of the Local Government Board has no more sincere friend and well-wisher than his present critic, for I am fully aware that he is whole-hearted in his anxiety to grapple with the scourge of consumption. Before leaving this matter, let me quote one sentence from the circular for which I am truly grateful. On p. 4, the secretary says: "It may be added that in structure sanatoria may be of the simplest and cheapest form. Wood and iron sanatoria have been provided at Woodilee and Garbeth asylums, at approximately £60 per bed. Open-air shelters can be provided at from £6 to £8 per bed, or even less."

In what remains of my time I wish very briefly to bring before you a few of the facts I ascertained by the inquiry to which I alluded at the beginning of this article.

My inquiries were directed to find out the number of beds available for the treatment of poor or assisted patients, the length of treatment found necessary, the demand for admission which existed, the stage of the disease when admitted, the style of building, altitude, site, and construction of the sanatorium, the staff employed, mode of government, the sources of income, cost of the structure, and of maintenance, and the results of treatment. I will only attempt to discuss a few of these points.

1. *The number of beds.*—In all I found that 1084 beds were open in July 1905 throughout Great Britain. All but two of the sanatoria were intended primarily for early cases, but some were compelled to admit a few intermediate ones. The length of treatment varied from a few weeks to six months. The average was three and a half months. About 3000 patients were treated annually. Now in 1905, 45,324 persons died from phthisis in Great Britain. There are no statistics to show what number of persons living are the subjects of the disease, but experts generally agree that they must be five times as numerous as the fatal cases.

The case mortality of phthisis is much disputed, being variously estimated by different authorities at from three to eight times the

number of deaths. The duration of the disease of the ordinary type is so long, probably five or six years on an average, that the figure I have mentioned cannot be excessive. The number of cases of incipient disease, before an assured diagnosis can be made, must be very large, judging from the post-mortem evidence I have already alluded to. They are likely to increase rapidly as the means of diagnosis become more extensive and accurate. The opsonin method and Calmette's ophthalmic reaction, as well as a more careful study of physical signs, may enable us to anticipate the appearance of lung invasion, and so enormously increase the number of those who ought to receive preventive treatment.

It is no exaggeration to say that in the year 1905 fully 200,000 people could be found in various stages of the disease. Suppose we reckon the early cases at a fourth of that number, we find that 50,000, of which at least 40,000 must be poor people, were in need of sanatorium treatment. You will agree with me that 1084 beds are grossly inadequate. From all of my correspondents came the pathetic tale of having to refuse hundreds of applicants for admission, from want of room. Since 1905 a good number of such sanatoria have been opened, and probably the beds available may now be 1600, with room for about 5000 patients annually.

2. *The sources of income.*—In all but four a considerable part of the income was derievd from patients who paid all or part of their board, the sums varying from 2s. 6d. a week up to £2, 2s. They were therefore enabled to meet the demands of middle-class persons who could not afford private sanatorium charges. The income so received helped to keep the institution open for their poorer brethren. Public and private voluntary contributions in the form of endowment of beds or payment for patients or ordinary subscriptions were received by all. Subscriptions from friendly societies were given in several cases, and in nine sanatoria help was given by the Town, County, or Parish Authorities, either as subsidies or endowment of beds, or payment for individual patients. In one (Durham County Sanatorium) a novel and most excellent system obtains, namely, a levy (voluntary, of course) on working men of 1s. a year, in virtue of which the subscribers are entitled to free treatment for the average length of time for themselves, or any member of their family over 15 years of age.

This yields about £700 a year, and has been satisfactory to all parties. The men receive also a share in the management of the

institution. The mode of government generally is to give due representation to all who support the sanatorium as far as possible in proportion to their contributions.

3. *The cost of the structure.*—On this point it was found impossible to arrive at an accurate or in some cases even an approximate estimate. Several were old buildings adapted and extended, and in two were merely rented. In six instances they were gifted by wealthy donors. These cost between them nearly £400,000, or an average of £760 a bed. The highest was £1000, and the lowest £620. Possibly, according to the ancient saw, we ought not to "look a gi'en horse in the moo'," and presumably these buildings have been erected in accordance with the terms of the donor, but it is certain that that sum is far too great for a working-class institution. Yet we find other sanatoria, perhaps in a spirit of rivalry, erected at a cost not much less, though drawing their funds from people who cannot afford such magnificence in their own homes. Two of them reach £500 a bed, and one £300, and the average of all the non-gifted institutions is £260 a bed. How much the glory of the promotors, or the ambition of the architects, or the greed of the contractors, have to do with this state of things we know not, but honesty compels us to call it extravagance. The furnishing and maintenance of these palatial places more or less corresponds with the style of architecture, and provides luxury utterly unsuited to the plain tastes and the proper needs of the inmates. At first they feel uncomfortable and out of place, and by and by may blossom out into "bloated aristocrats," discontented with the humble home to which they may be able to return. We hope they may for ever be disgusted with dirt and evil odours, but not with simple food and plain furnishing. What do they want with tiled hearths, silver-plated fittings, stained-glass windows, and gilded decorations? Beauty is all very well, but surely it is out of place in such a building. Economy is absolutely necessary if we hope to carry the public with us in this crusade.

We can find such economy in a few of the sanatoria, and it is noteworthy that their record as curative institutions is as high as the extravagant ones. Durham Sanatorium is a case in point. The new wings it added to the old building, which was a mansion-house, and is used as an administrative block, cost only £80 a bed. Also at Kelling, in Norfolk, the cost was only £130 a bed. At Kelling two pavilions have just been erected of "Mack" slabs at

£59 a bed. Both these sanatoria have an excellent record of recoveries. At Woodilee and Gartloch the buildings cost only £90 a bed, and every essential is to be found without any unsightliness or discomfort. At Craiglockhart we have a building of the same unpretentious but efficient character.

On the Continent and in America, buildings of a still simpler and cheaper kind have been used with complete success, and the shanty or shelter referred to in the L.G.B. circular has been extensively used in the grounds of sanatoria, as at Craigleith and many other places. If the money which has been spent on the structure of sanatoria in Great Britain up to this time, and which must have amounted to three-quarters of a million at least, had been used as it might have been, more than 7000 beds could have been now available, and 20,000 patients received treatment.

4. *Cost of maintenance.*—The management expenses of the sanatoria varied in a surprising way, though not so strikingly as the capital cost. In two it reached as high as £2, 2s. a week, and in one case sank to the modest sum of 10s. a week per occupied bed: the average over all being about 28s. a week.

In an institution in which good feeding is an essential part of the treatment, one would have expected this item of expenditure to be fairly constant, but it was nearly as varied as the general expense. The food bill stood at 20s. a week per occupied bed in one instance, and at 6s. in another. No difference in locality, or price of commodities, could account for such discrepancy. Home produce in the shape of vegetables, fruit, milk, butter, eggs, poultry, etc., no doubt reduced the cost in some. With three exceptions, and even then with modification, the now discredited over-feeding system has been abandoned, and a generous diet substituted. But there can be no doubt that a dainty fare, for which there is little excuse in the case of people used to simple diet, must ye responsible for some of this extravagance. The average food bill stands at 11s. 6d. per week, and if we exclude some of the sanatoria as extravagantly provided for, the average would stand at 10s. per patient, or even less.

In connection with this maintenance question, it is reasonable to compare sanatoria with other similar institutions. The district asylums of Scotland run them close in matters of staffing, in management requirements, and in the class of patients received. The average cost of maintenance in 1904 was 10s. a week per occupied bed, of which 4s. was spent on food. Admitting that

this latter amount could not provide a sanatorium diet, and adding 7s. 6d. to bring it up to the standard, we found the total management should be 17s. 6d. in place of 28s. per week. In the Edinburgh Royal Infirmary the total cost of maintenance for an average of 832 patients, and 400 resident members of the staff, amounts to 24s. per occupied bed per week and the food bill to 4s. 4d. for the patients and 7s. for the staff. The cost of drugs, instruments, dressings, etc. amounts to 2s. 6d. a week per patient and, as they are an infinitesimal expense in sanatoria we may claim that hospital maintenance stands at 21s. 6d. as compared with 28s. The Aberdeen Royal Infirmary stood at 20s. per week in 1904 and the food bill at 7s. There is emphatically room for economy in sanatorium management, and if we expect the public to be with us in the crusade, this must be attained.

5. *The results of treatment.*—This point need detain us but a moment. The results are so differently reported that they cannot be tabulated. In some cases the after-history has been traced to from one to five years. Without attempting to go into details, I have no hesitation in saying that 60 per cent. of the early cases must have been arrested or temporarily "cured," though that word is not an appropriate one for any disease of a chronic nature if used without qualification. After some years watching 40 per cent. of them still maintained their health and capacity for work. Even a proportion of the intermediate cases were restored to health and usefulness, varying from 10 to 15 per cent. In almost every case an immense benefit was derived from the treatment, enough to make any institution carried on under secret methods, and run on profit bearing lines, a mine of gold to its proprietors. Even if this were all, and only a few months of relief, or a few years of life, were the result, would any of us grudge all he possessed to purchase a respite from the approach of the last enemy, or fail to bless the hand that shielded us even for a time from his assault?

But even if this were all, and the "cures" as illusory as some arm-chair critics would have us believe, the sanatorium has justified its existence as an educational institution of the first water. From it come forth men and women who have been taught the principles of healthy living, and, as "living epistles known and read of all men," preach a gospel of health far more effectively than an army of inspectors or tons of literature.

In conclusion, and to sum up, let me repeat the salient points I have tried to bring out—

1. That phthisis is a disease that can be cured by open-air treatment in the early stage.

2. That the poorer classes are the chief sufferers, and that we are bound to help them on humanitarian and even selfish grounds.

3. That curative treatment cannot be a matter of State intervention, but a duty of the whole community, including the poorer classes themselves.

4. That strict economy in providing and maintaining sanatoria is essential in enlisting public sympathy and support.

3. OBSERVATIONS ON THE USE OF INOCULATIONS OF BACTERIAL SUSPENSIONS.

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Immunity against bacteria may be of various kinds, namely, antimicrobial, that induced by the receptors of the bacterial bodies themselves, and antitoxic, that induced by the soluble toxins produced by the bacteria and given off from them, or possibly also by those retained within them as endotoxins. The processes involved are essentially different, and at present we are concerned solely with the former, *i.e.* antimicrobial immunity. For its production we would wish to inject nothing of a toxic nature but the bacterial bodies with their contained receptors. The protoplasmic bacterial poisons (receptors) possess specificity within certain limits dependent on their stereochemical structure which is sometimes easily upset by physical and chemical means. In practice the bacteria from cultures are first killed, usually by heat, washed, and injected in suspension in a neutral fluid.

If one introduces a minute quantity of poisonous bacteria into the system, the organs react to protect themselves, the reaction is enormously out of production to what is required to neutralise the poison introduced, and the protective substances formed are therefore available for combating existing disease or guarding against future infection of the same nature. It is important to realise fully that one is not introducing into the patient's system

a dose of a ready-formed protective or anti-substance or serum for one is relying on the patient's immunising reaction to produce this for himself, and if he were unable to do so, such treatment would be unjustifiable.

One may naturally wonder why such inoculation can be beneficial in the presence of an infective disease. The reason is to be found in the observed facts that nature tends to control infective processes as far as possible by localising them and preventing their extension to the general circulation and distant organs. In this work first the leucocytes, later the fixed tissue cells, play the part of the limiting agents, with the result that where the local reaction is efficient the organisms are early shut off from the circulation, and sufficient general stimulus is apparently wanting. The tendency later is to throw out the infective agents from the body, and so we have in some instances the rupture of abscesses and the formation of granulation tissue, which presents an excreting, not an absorbing surface.

If the barriers are incomplete, then the stimulus reaches the body circulation, and in such cases inoculation may be superfluous if not actually prejudicial.

In purely localised cases we can produce and increase by inoculation the quantity of specific antibacterial substances in the circulating blood plasma. The local lesion is benefited in direct proportion as this plasma reaches the organisms and injures them or prepares them for phagocytosis. A marked positive chemotaxis is induced, attended probably with increase of local inflammatory reaction. At any rate one finds that where suppuration has already developed, it tends to be augmented for a time and to terminate more rapidly. If a discharging lesion such as an open abscess is present, the effects can be demonstrated by microscopical examination of the pus. Before inoculation one finds perhaps organisms in quantity lying free but rarely within the leucocytes, after inoculation but few extracellular organisms and many leucocytes packed with them. When suppuration is concealed at first, the pus when evacuated is not infrequently sterile. The blood plasma after inoculation is seemingly able to supply just that needful help to the leucocytes which are no doubt poisoned by the products of bacterial growth, those non-specific toxic substances such as the tox-albumins common to all organisms causing suppuration, and the outcome is that the tissues finally gain control of the situation.

This is the typical sequence of events in lesions due to the bacteria of acute suppurative infections. With others, *e.g.*, tubercle, however, the case is different, where the resulting lesion is a very chronic one, and so formed that the bacteria may be immured in it and cut off from reach of blood and lymph. Inoculation here has probably little effect directly on the bacteria, but if the blood is rich in protective substances, extension of the disease is unlikely, for any bacilli escaping from their confines are bathed in this fluid, and phagocytosis is actively induced. If we can control the extension of any chronic disease, we are doing a great deal of good to the patient, and usually local factors will in the end eliminate the local infection. In the very nature of things, then, inoculation treatment in such conditions as tubercle is less striking in its results, but hardly less valuable. Of course the logical endeavour in inoculation treatment is to increase as far as possible the circulation of the immunising plasma through the diseased area. Accordingly heat, and appliances for producing hyperæmia, are of great value, and should be adopted where feasible.

Till recent times inoculation treatment was to a large extent empirical, and with doses given in regularly increasing amount and at regular frequent intervals, aimed at "accustoming" the patient by degrees to large quantities of the poisons from which he suffered, so that they in their turn should prove harmless. At first an obvious clinical reaction was aimed at, subsequently the aim has been to avoid this. In recent times, Wright and his school have combated this method of treatment, claiming that the real object is to increase the protective substances in the blood of the patient, and that this must be controlled by the observation of his phagocytic index.

I must here digress to refer briefly to the effects of inoculation as shown by a study of the index. If a dose of a bacterial suspension within therapeutic limits is given to a healthy person, the result is a rise in the index, usually showing itself at the end of twenty-four hours, then progressively increasing for a variable period, and lasting altogether for several weeks. In an infected person this rise or "positive phase" is preceded by a temporary fall or "negative phase" (see Chart 1). What is not so generally known is that if the index is observed at short intervals within the first twenty-four hours a very marked rise occurs, usually attaining its climax in about six hours, and this is a

feature common both to healthy and to infected persons' bloods (see Chart 2). This observation is of importance as showing that the infected person is not less capable of rapidly elaborating immune substances.

Wright and his school insist on the importance of avoiding giving an inoculation during a negative phase or period of supposedly diminished resistance, on account of the risk of producing a cumulation of negative phases, and consequently

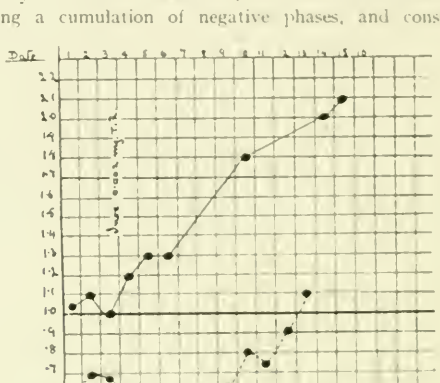


CHART 1.—Showing effects on day to day indices of a healthy and an infected person after inoculation. The healthy person's blood (continuous line) shows an immediate positive phase; the infected person's blood (broken line) shows a preceding negative phase.

abolishing the powers of resistance. This was observed by Wright to occur with regard to the *agglutinins* to *B. tuberculosis* in the blood of a patient treated by frequent inoculations of tuberculin, whose agglutinative power fell to zero. He apparently assumed that the same would occur in respect of the phagocytic index, but so far as I know it has not been actually tested and reported on. My own observations do not bear this out. It is on this assumption, however, that his method of controlling inoculation treatment is based. It should be realised that the phagocytic index represents only a limited phase of immunity, and that proof

is wanting as to the prejudicial significance of the negative phase as such.

Phagocytic index estimations to my mind have proved their greatest value in showing how small a dose of a suspension is necessary to produce a marked immunising effect, in showing how long such effects may persist, and in showing the fallacy of the

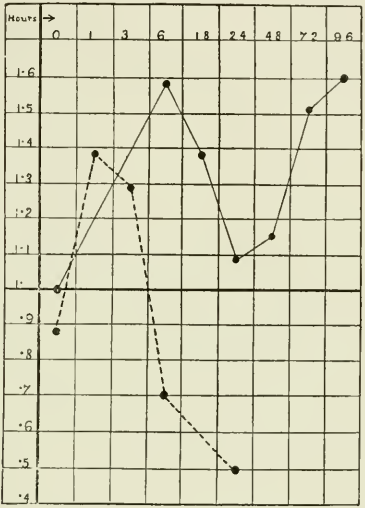


CHART 2.—Showing early effects on indices of a healthy and an infected person after inoculation. The bloods both of the healthy person (continuous line) and the infected person (dotted line) show a marked positive phase within six hours. At the end of twenty-four hours, however, the position corresponds to that shown in Chart 1.

frequent use of inoculations, for it is found impossible to call forth a progressively cumulative and lasting positive phase in a patient's blood. If this is attempted, one finds after a number of inoculations, each given while the index is still high, that a negative phase may occur, which is much prolonged and may last for weeks.

On the other hand, I have found that with a suitable dose, if each successive one is given only as the effects of the previous one are passing off, the treatment may be continued for months

or years without incurring the risk, if such it is, of a prolonged negative phase. In this procedure the index, without perhaps reaching any great height, is maintained very steadily above normal. If we can secure this we shall probably succeed in preventing any extension of disease in those very chronic cases which demand long-continued treatment. My experience has convinced me that the practical results are as satisfactory and in the long run more so than if one aims at a supposedly more rapid method of immunisation. Nor is there probably any real advantage in attaining a very high index, as that only demonstrates the presence of phagocytic immune substances in the circulating blood in great excess of what are absorbed by the disease focus, which are therefore superfluous.

Except in the case of tuberculin, it has been the custom to measure bacterial suspensions in terms of the number of organisms contained. This precludes any comparison of dosage of different bacteria. It has been my practice to prepare all suspensions by weight, and measure their dosage in terms of the weight of washed and dried bacterial bodies contained per c.c. This corresponds to the description of dosage in Koch's "new" tuberculin preparations. The result has been that one could compare the dosage of different bacteria, and I have satisfied myself that doses approximately equal of all bacteria are correspondingly efficacious. It is generally held that a dose of Tuberculin-R. need not exceed $\frac{1}{1,000}$ mgrm., while a dose of 1000 million staphylococci, which weigh as much as $\frac{1}{10}$ mgrm., is considered quite an average dose, though weight for weight a hundred times that of the tubercle bacilli. A dose of $\frac{1}{10,000}$ mgrm. is an amount which I have not found it necessary to exceed with any bacterium with which I have worked, and the results are as satisfactory, I believe, as with hundred times larger doses commonly given.

An objection that might be raised to this method of weighing bacteria for suspensions is the larger quantity required and the consequent delay if prepared from the patient's own bacterium. This is held to be necessary in many infections, typically streptococcal ones, the idea being probably based on the supposed necessity of producing polyvalency in antistreptococcal serum preparation. This is quite contrary to my own experience, as I have not found any infection fail to beneficially respond to the use of a stock suspension.

But in order to obtain a bacterial suspension which is in itself equivalent, certain points must be observed in its preparation. The bacteria must be freshly isolated from a source of active infection, the cultures must be young and grown on the most suitable media, and subcultures should be avoided if possible. In short, the cultures must preserve the highest degree of potential pathogenicity. They are then washed entirely free from cultural by-products.

The proper intervals as indicated by following the phagocytic index would be found to vary to some extent at different times and in different individuals. We wish to anticipate the fall of the index below par, and we shall probably do this in every case by giving injections of suitable amount every four weeks. That is perhaps oftener than every case would require, but we need not fear doing harm.

A few cases show a markedly persistent negative phase after a first inoculation. This is possibly due to a somewhat obscure feature of immunisation known as hyper-susceptibility. It is overcome, and a positive phase produced by giving another inoculation without further delay. I therefore feel inclined to recommend giving two or three inoculations close together at the beginning of treatment. A simple scheme then for treating a chronic condition in an adult would be to give doses of $\frac{1}{1000}$ mgrm. at intervals of two, then three, then four weeks, continuing at that rate for six months. Later, probably, it is not necessary to inoculate oftener than once in six weeks, and finally perhaps in two months. But this later treatment must depend on the clinical condition and the extent of recovery. Without exception, inoculations in chronic affections, typically tubercule, should be continued long after recovery is assumed, to prevent relapses.

In children under 14 the dose is reduced to one-half, and progressively with diminishing age, so that the dose for infants is perhaps one-tenth.

More acute conditions demand smaller dosage, and where prolonged treatment is not anticipated, inoculations may with advantage be given at shorter intervals, so as rapidly to bring the patient under the greatest possible influence of the drug. For example, in acute infections a dose of $\frac{1}{1000}$ to $\frac{1}{2000}$ mgrm. is given and repeated in five to ten days.

I have treated now very numerous cases on these lines, and the results have been quite satisfactory. The guiding rule should

be kept in mind that when in doubt it is better to withhold than to give an inoculation.

As regards the method of administration, I have found the left triceps about 2 to 3 in. above the tip of the olecranon, the most suitable site for injection. The suspension is injected direct into the muscle. It should be made up with normal saline, glycerine being avoided, and after the initial sterilisation preserved by the addition of a few drops of chloroform. Given in this way no discomfort ensues after inoculation.

It would be impossible for me here to review in detail the whole scope of inoculation treatment. I shall confine myself to a few remarks on the conditions with which I happen to have had most experience.

Firstly, with regard to *tubercle*. Most surgical affections are of the localised chronic type. If fever is present, in the absence of mixed infection, it is an indication of absorption from the disease focus, and rest is necessary till it subsides. The case is then treated as a localised one. Mixed infection demands treatment with the infecting organisms as well as with tuberculin The rapidity of improvement is then often surprising.

Gland affections show very variable results, depending on the stage of disease. Very early cases do well. Caseating glands are very slow of absorption, and only time will accomplish this. Operation is often advisable, then, to hasten complete recovery. Rapidly breaking down glands often do particularly well, and usually require only puncture, repeated if need be, to evacuate the fluid. Periadenitis rapidly disappears with two or three injections, and leaves the glands becoming small and firm and much better suited for operative treatment.

Bone and joint cases give excellent results if not too far advanced. Infected joints, if got early, may recover with no loss of mobility. In any case restriction of movement may be less rigidly or continuously applied under tuberculin control, and while recovery with mobility is so important in the upper limb this may also be aimed at in the lower. Discharging sinuses, if not actually leading to sequestra, can as a rule be got to heal, but mixed infection must be treated if present.

Skin conditions are specially intractable unless tuberculin is combined with suitable local measures. Here especially, as in most tuberculous affections, congestive treatment is of value.

We are all familiar with the cases of multiple infection, such

as those patients "eaten up with tubercle" who haunt the out-patient departments of hospitals. A study of many such cases has led me reluctantly to the conclusion that too frequently operative procedures have been but the starting-point for a fresh extension of infection. In some instances, indeed, one has found that each operation has been regularly succeeded by a new appearance of tubercle elsewhere. Here we have a suitable field for the prophylactic use of tuberculin. So convinced have I become of this risk of extension, which, though not by any means the rule, cannot well be foreseen, that I would not readily bring myself to operate on a case of tubercle which had not been "protected" by a preliminary series of tuberculin injections.

The possibility of a certain number of cases of tuberculosis being due to bovine tubercle, as I have shown elsewhere,[1] must be kept in view, and before carrying out any prolonged treatment, one of the diagnostic methods which have been published from the University Surgical Laboratory[2] may be employed, and the patient then treated with a suspension of the human or the bovine type of bacillus, as determined by the blood examination.

The general surroundings and mode of life of the patient call for most careful attention, for if not favourable it is probably not advisable to use tuberculin.

Staphylococcal infections, so common in surgical work, offer a large field for treatment. Abscess formation may be prevented in the early stages. If already developed, healing is greatly hastened. Boils, a frequent cause of much suffering, can be aborted. If a slough has already formed, it usually separates in two to four days after inoculation, and healing follows as rapidly. Relief of pain is very prompt with a suitably small dose. Recurrent crops of boils can be entirely obviated once treatment is carried out, but this should be prolonged after recovery when the condition is a chronic one. Acne is similarly amenable to inoculation, but as the inflammatory condition is only secondary, the primary skin affection must certainly not be neglected.

In *streptococcal* infections I have seen more strikingly good results than in any others. Streptococci are responsible for most diverse types of disease, highly acute or intractably chronic. All are amenable to treatment. Using a single stock suspension prepared from a case of wound infection, I am the more surprised

[1] Ritchie, *Brit. Med. Journ.*, London, 1907, November 16, vol. ii.
[2] Stewart and Ritchie, *Edin. Med. Journ.*, 1907, May. Ritchie, *loc. cit.*

at my uniformly good results, for it is generally claimed that streptococci for injection must be prepared from the individual patient. Such has certainly not been my experience. Acute lymphangitis, wound infections, chronic sinuses, erysipelas, have all yielded equally well to the one preparation. This is a fact of some importance, as many streptococcal infections spread so rapidly that time is not given for the preparation of a suspension. Acute lymphangitis spreading up a limb, if treated early, is usually gone in twenty-four hours. Pain is relieved in a few hours, often completely in six; the accompanying malaise passes off at the same time, and the patient probably obtains an undisturbed night's rest. This course of events I have seen time and again, and it is not influenced by omitting local treatment. It is specially interesting as showing how the clinical condition corresponds to the early high positive phase which I have shown you on Chart 2. As an instance of an opposite type of case, I may mention a 6-in. long, chronic sinus following on empyema, infected with streptococcus, which reopened and discharged for six months, till after a single inoculation it healed permanently in less than a fortnight.

I do not propose to refer to other infections in which inoculation treatment has been tried and proved successful. Suffice it to say that the general principles stated are equally applicable to them.

Finally, I wish to refer to the prophylactic use of bacterial inoculations. From my previous remarks you will understand how one can assume the prophylactic action of immunising substances to be more efficacious than the curative. In the case of such a non-vascular lesion as a tubercle, treatment is essentially prophylactic; we guard against extension, and leave largely to local processes the final extinction of the disease.

But prophylactic treatment in the ordinary sense of anticipating infection has enormous possibilities. Here, however, we are faced with the difficulty of not always knowing what organisms we shall wish to be able to combat. The difficulty in the case of wounds, operative or accidental, is more apparent than real. If we give a dose of $\frac{1}{1000}$ to $\frac{1}{100}$ mgm. each of *Staphylococcus aureus* and *Streptococcus pyogenes*, and also of *Bacterium coli* if in the region of a mucous tract we shall probably cover most eventualities, for other infections are usually secondary. I give such an inoculation in all operation cases, with so far uniformly satisfactory results. In accident wounds, too, we

can similarly control if not entirely prevent suppuration. The early positive phase occurring within the first few hours, and the absence of subsequent negative phase in healthy persons, as already mentioned, furnish the justification for such inoculations given at the time of operation or soon after an accident, as any growth of bacteria in the wounds is met by a blood fluid already rich in protective substances.

Numerous cases have been treated thus prophylactically in the Surgical Out-Patient Department of the Royal Infirmary, and it has proved very valuable in aiding the conservative treatment of lacerated wounds, in primary skin-grafting, and generally in all cases where complete wound disinfection is difficult to ensure.

It should be noted that if this method is employed, the use of chemical antiseptics in the tissues is contra-indicated, as these inhibit phagocytosis, which is the means of final disinfection on which we are relying after the necessary mechanical cleansing.

I have pleasure in once more recording my thanks to Professor Chiene for the facilities accorded me in his Laboratory for the experimental work on which these observations are based, and I would likewise gratefully acknowledge the kindness of the surgeons in charge of the Surgical Out-Patient Department of the Infirmary, who have so generously afforded me the opportunities for the study of inoculation treatment.

Special Meeting.—March 18, 1908

Dr James Ritchie, *President, in the Chair*

Original Communications

1. RECENT RESEARCHES INTO THE ETIOLOGY OF PLAGUE IN INDIA

(With an Account of the Work of the Plague Commission recently appointed by the Government of India, whereby it is shown that The Rat Flea is the Cause of Plague Epidemics in that Country. With Lantern Illustrations)

By Lieut.-Colonel W. B. Bannerman, M.D., B.Sc., F.R.S.E., Indian Medical Service; Director, Bombay Bacteriological Laboratory; Associate Fellow, College of Physicians, Philadelphia; Corresponding Member, American Society of Tropical Medicine

The Plague Research Commission, of whose work I am to give an account to-night, was appointed by the Right Hon. the Secretary

of State for India, acting on the initiative of the Government of India, who had moved in the matter in the autumn of 1904. To assist in the guidance of the work an Advisory Committee in London was appointed by him in consultation with the Royal Society and the Lister Institute of Preventive Medicine, and was composed as follows: Surgeon-General A. M. Branfoot, C.I.E., I.M.S. President of the Medical Board, India Office, *Chairman;* Sir Michael Foster and Dr J. Rose Bradford, representing the Royal Society; Colonel David Bruce, C.B., R.A.M.C., and Dr Charles J. Martin, representing the Lister Institute, *Members.* After the death of Sir Michael Foster in January 1907, his place was taken by Dr G. H. Nuttall, F.R.S.

This London Committee appointed a Working Commission in India as follows: Major G. Lamb, M.D., I.M.S., Director of the Pasteur Institute of India, Kasauli, *Senior Member;* Captain Wm. Glen Liston, M.D., I.M.S., Assistant to the Director, Bombay Bacteriological Laboratory; Captain T. H. Gloster, M.B., I.M.S., formerly Port Health Officer, Rangoon; G. F. Petrie, M.D., Assistant Bacteriologist, Lister Institute; Sydney Rowland, M.R.C.S., L.R.C.P., Assistant Bacteriologist, Lister Institute; Assistant-Surgeon M. Kassava Pai, M.B. (Madras); V. L. Manker, L.R.C.P. & S. (Edin.), D.P.H. (Camb.); Hospital-Assistant P. S. Ramchandrier, and Hospital-Assistant C. R. Avari. It will be noticed that, with the exception of the two bacteriologists sent out from the Lister Institute, the personnel of the Commission was connected with the medical services of the Government of India, and that four were natives of India. This fact should prove of interest to politicians in these days of travelling Members of Parliament.

The Commission had its headquarters in Bombay, and was given laboratory accommodation at the Bombay Bacteriological Laboratory at Parel. Dr C. J. Martin, the Director of the Lister Institute, went to India in the end of March 1905, and organised and started the work at Parel, after satisfying himself, by personal inspection of the other Indian laboratories, that, on account of the presence there of a trained and organised laboratory staff and ample supply of animals, it was the only place in India where work on the scale contemplated could be carried out. He returned to England after six months, and since then the head of the Commission has been Major G. Lamb, I.M.S.

The major part of the experimental work was carried out in

Bombay city, but most essential investigations were, in addition, conducted in two small villages in the Punjab, where facilities existed for watching an epidemic with some approach to accuracy.

At this point it will be instructive to consider the extent of our knowledge of plague as derived from Indian experience, at the time the Commission arrived in India.

The present manifestation of plague in India dates from the autumn of 1896, when the first case was diagnosed by Dr Viegas, a well-known medical practitioner in Bombay. We knew that plague appeared in the remote valleys of the Himalayas in the region of Kumaon, in sporadic form from time to time, and that the people there deserted their villages on its approach; but that it had any practical significance for the medical man in India no one imagined. It was regarded as in the nature of an antiquarian curiosity belonging to these old-world regions, and as for ever banished from the civilised country of Hindustan. It followed, then, that the average practitioner in India knew nothing about the disease when it invaded Bombay. Warnings had indeed been issued by Colonel W. G. King, C.I.E., I.M.S., the energetic Sanitary Commissioner for Madras, to all Government Medical Officers in that presidency, to look out for its importation from China, where it had broken out in epidemic form in the year 1894, but it is not matter for surprise that some time elapsed before the local medical faculty recognised the dread disease or realised its significance. Still less were they in a position to take prompt steps to stop it, or to advise correctly what the nature of these measures should be. Within a fortnight, however, of the recognition of the disease, the Government had sent Mr Haffkine to Bombay from Calcutta, and had convened a Committee of local medical men, with the Professor of Medicine in the Grant Medical College, Bombay, as President, to consider what should be done. I mention these facts merely as a mild protest against the recent assertions of certain writers in the medical and lay press, who appear to judge of the action or inaction as they are pleased to term it, of the Government of India, by the light of knowledge acquired by ten years of laborious research carried out after this period.

One of the first things that our practical experience of plague in the Bombay hospitals revealed, was that there are two distinct types of plague; one, the pneumonic, which was very contagious, and the other, the bubonic and septicæmic, which was not communicated from one person to another. To our surprise, we found that

the attendants in hospitals and the friends of patients who accompanied their sick to the wards did not take plague unless there happened to be a pneumonic case in the institution. In fact, so marked was this, that Major Thomson, I.M.S., who had charge of the large hospital opened in the Old Government House at Parel, reported "that one of the safest places during the epidemic is the ward of a sanitary plague hospital, something more than mere contagion being necessary to develop the disease" (Gatacre, 1897, p. 52).

Another fact that soon became apparent was the curious seasonal prevalence of the disease. It was found that each zone of country had an epidemic season of its own, each place having its epidemic at its own particular time and never at any other.

Thus we find the epidemic in Bombay city takes place in the early months of the year, while at Poona, a town only eighty miles away, the disease does not appear till autumn, when Bombay is almost free. In other words, the plague appears in a place, increases in virulence and extent, declines and disappears, on or about the same date annually, but the time of year varies with each locality.

As years passed and epidemic succeeded epidemic, facts came to light which pointed to huts or houses becoming infected with plague and remaining so even after the inhabitants had fled.

Such rooms were found or suspected to be most dangerous at night. Over and over again cases of infection in Health Camps were traced to surreptitious visits paid to houses in infected villages, so that those responsible for the administration of these camps, came to regard "dropping cases" of plague as proof of the inefficiency of their village cordons, for investigation generally led to the discovery of secret visits to the abandoned houses at night. Again, the occurrence of unexpected and heavy rain, which compelled the people to abandon their flimsy huts and return to their houses, has often been followed by a virulent outburst of plague amongst them, though they had remained free from the epidemic whilst living in camp (Condon, 1900, p. 23).

It was found even that houses might become infective while standing empty. When it became common for people to quit their houses because dead rats had appeared in them, instances came to light where a community had abandoned a village or quarter before plague appeared, and yet on their return, after an interval of some weeks, a severe epidemic would break out, appearing simultaneously in several distinct quarters of the village. Evidently

the houses had become infective during the absence of the owners (James, 1899, p. 97).

The whole of the above evidence went to prove that man-to-man infection was not the cause of the spread of plague, but that we must look elsewhere for the active agent.

From the very earliest times it has been a common observation that rodents became affected in a peculiar way during plague epidemics (*vide* 1 Samuel, chaps. v. and vi.), but it was not till Yersin and Kitasato announced the discovery of the *Bacillus pestis* in rats and mice in Hongkong in 1894, that it became certain that the disease in question was really plague. In India these observations were confirmed by Haffkine and Surveyor in October 1896, *i.e.* the very month in which plague was diagnosed with certainty in that country. Overwhelming evidence very soon came to light that rats spread plague in India.

It is impossible in the present paper to give more than a brief outline of the evidence in this direction. Those desiring further details are referred to the voluminous evidence placed before the first Plague Commission (which, under the presidency of Sir Thomas Fraser, visited India in 1898-99), and published by them in five monumental Blue-books in 1901; or, for additional and more recent instances, to a paper by the present writer entitled, "The Spread of Plague in India," published in the *Journal of Hygiene*, vol. vi., 1906.

It was early noticed that houses in close proximity became infective, though their inhabitants, owing to caste or other prejudice, could have had no communication with one another. Cases like the following could be multiplied (James, p. 96). A rat was seen by a Sikh family to come out of a hole in the wall, to wander about the room in an aimless sort of way, and shortly to die. A day or two after plague breaks out in this house, and it is found on investigation that the rat-hole leads into a neighbouring house where plague had occurred a day or two before. No communication could have taken place between these two families, for they belonged to castes that are abhorrent to one another. Again, two families are attacked in a Punjab village, one a Hindu, the other a Muhammadan family. The first case took place in the Muhammadan household, who lived in a courtyard just outside the village. To enter the Hindu's house, it was necessary to traverse tortuous lanes leading apparently to the centre of the village, but on mounting to the flat roof it was found to be

separated from the Mussulman's house by a small cowshed only. When the practice of unroofing houses for purposes of disinfection came into fashion, it was easy to trace the course of the disease through the villages by mounting to the roof and noting the lines of unroofed huts to be seen. These could be seen stretching in continuous lines through all the different quarters of the town, irrespective of the caste of their occupants.

These and similar examples irresistibly suggest the idea of spread by animals whose runs pass from house to house irrespective of caste or other prejudice, and the only animal that fills the role is the house rat of India.

Another fact that came to light, as our experience accumulated, was that the epizootic precedes the epidemic.

Examples of this are to be found in abundance in the volumes of the first Plague Commission Report, or in the annual reports of medical officers on plague duty, but as it is nowhere so clearly shown as in the chart for the city of Bombay for the year 1905-06, I refer to it here, though this was compiled by the second Plague Commission, and will be noticed in detail later on in this paper, (*vide* p. 181, *infra*). Suffice it to say that dead rats are looked upon as the precursors of plague, and the rule, "When dead rats are seen, clear out or be inoculated," is now almost universally acted on throughout India.

All our Indian experience, then, points to the rat as responsible for the spread of plague, but is it wholly responsible?

That plague can in some way be communicated both to men and rats by infected clothes is a common belief in India. Instances like the following, related by Major Collie, I.M.S. (1898), are common. "A man lost his wife from plague in Bombay, and ten days later he brought her clothing and ornaments to his house in a village near Harnai, in the Ratnagiri Collectorate. In about a week dead rats were found in this man's house and neighbourhood, when one relative after another sickened and died from plague, and ultimately the man himself became the sixth victim. None of his relatives had been out of their village." This was the first outbreak of plague in that part, and no other means of importation could be discovered. Again, it became a common practice in the Punjab for people to send away their best clothes to friends in distant villages, so as to avoid the necessity of disinfection, which they feared might deteriorate them (James, p. 86). In this way many infections of outlying villages are believed to have been brought

about. It did not necessarily follow that the person bringing the clothes took the plague, for more often, perhaps, the disease first appears among the rats in the vicinity, and only afterwards among the inhabitants. After an experience of five epidemics in the Punjab (Wilkinson, 1904, *a*, p. 29), we find it reported that the sweepers of the Ambala district consented to the burning of all the clothes of persons dying of plague, instead of taking them away as perquisites, as had formerly been the custom. In Patiala state, these menials refused to touch either bedding or clothing of plague patients, which in consequence had to be burned where they lay. Major Wilkinson, I.M.S., also reports that "several instances were observed where thieves who had stolen infected clothing and taken it away to their own villages, died there of the disease" (1904, *b*, p. 17).

To sum up, then, we may say that Indian experience leads to the following conclusion. As regards introduction into a new area remote from a focus of disease, human beings are the carriers of infection, and every one agrees with the dictum of the Collector of Thana, who said, regarding this matter ("Rep. of San. Com. with Govt. of India for 1897," p. 190, 1899), that "plague adhered to the railway, and it almost confined itself to the area traversed by the local trains. Given accommodation for high-caste natives, and easy distance from Bombay, infection was everywhere a certainty." Within towns and villages, however, the case is far otherwise, for here the universal testimony is that rats are the means of the dissemination of plague.

The question then naturally arose, How does plague spread from rat to rat and from rat to man, and how can clothes be a means of carrying the disease from one place to another? The possibility of insects playing some part in the matter, of course, occurred to many. If, as the first Plague Commission believed, infection is always conveyed through the skin, it is evident that *biting* insects must be the criminals. In India, Simond of the Pasteur Institute was the first to suggest the flea as the carrier of the disease. He made a few experiments on this point, and was able in two instances to infect a rat kept in a glass case along with another rat sick of plague, actual contact being carefully avoided. He maintained that this theory explained much that was puzzling in the epidemiology of the disease, and hazarded the opinion that the actual mode of infection was from the fæces of the insect, which he noted were discharged upon the skin while the flea was sucking, and might easily be rubbed into the flea-bite in the process

of scratching. The first Plague Commission, however, adversely criticised his work, and considered his process of induction "so weak as to be hardly deserving of consideration."

They further conclude from the experience of plague hospitals in India, "that suctorial insects do not come under consideration in connection with the spread of plague" (vol. v. p. 77). When we consider the authoritative character of the first Plague Commission, and the eminent scientists who made up its personnel, it is little wonder that such expressions should have diverted the attention of workers in India from the view that fleas could have anything to do with the spread of plague. All the more honour then to my friend and colleague, Captain Glen Liston, I.M.S., himself one of the assistants to that Commission, for having seen the possibilities that this theory held out, and for having diligently collected facts, and, in spite of frequent discouragement and non-success, conducted experiments in the laboratory, which, by the time the second Plague Commission arrived, had placed the despised hypothesis in quite a new light, leading them to adopt it as that most likely to lead to success.

He began by studying the species of rats met with in Bombay, and the ecto-parasites infesting them. He found that there were two species of rats common in Bombay, both of which suffered from plague to much the same extent.

The one—*Mus rattus* (Fig. 1)—is identical with the black rat of England, which has become quite rare in Britain, as it has been overpowered and driven out by an invasion of the brown Norway rat (*Mus decumanus*), which, perhaps luckily for us, has become the common rat here. It is easily distinguished by its long, ringed, dark-coloured tail, which is *longer than the head and body together*. Its ears are large and elegant. The colour is very variable, the commonest shade in Bombay being brown, but all grades between that and black are found. The belly is invariably of a lighter shade, and when this is very marked, the animal has by some been classed as a variety under the name of *M. alexandrinus*.

This rat, by consorting for centuries with man, has become almost a domestic animal in India, and takes the place occupied by the cat in our houses, living as it does in every room, and even breeding there among the neglected rubbish, or in drawers and cupboards seldom opened. Being a most nimble climber, it lives largely in roofs and shelves, though it is capable of burrowing in earthen floors in gram stores and the like. It is largely a grain-feeder, and its nests are to be looked for in stacks of firewood

FIG. 1.—*Mus rattus.*

FIG. 2.—*Mus decumanus.*

FIG. 3.—*Nesokia bengalensis.*

From Photographs by C. R. Avari.

Fig. 1 is unfortunately on a smaller scale than the others.

PLATE III.—TO ILLUSTRATE DR W. B. BANNERMAN'S ARTICLE.

old bedding, or other accumulations of rubbish. To an animal of such habits, an Indian village, with its mud and thatched roofs to nestle in, and its stores of grain to feed on, must offer every attraction, and we need not be surprised to hear of 200 and more being found dead in a single house.

The other common rat of Bombay—*Mus decumanus* (Fig. 2)—the brown or Norway rat, has the *tail shorter than the head and body together*, and its ventral aspect is of a lighter shade of colour than the dorsal. The ears are smaller than in *M. rattus*, and the nose might almost be called Roman. The colour of the fur is brownish-grey, and much more constant than in *M. rattus*. It is not indigenous in India, but is now found in all seaports, having no doubt been introduced by shipping. It is not strictly speaking a house rat, though its extensive burrows are found at times in the lime-concrete flooring of the ground, and even of the first storey of the Bombay city houses. It delights mainly in municipal dust-bins and the garbage in the surface drains of the narrow gullies to be found between every block of houses in Bombay. It breeds in the extensive burrows it makes under the ground floors of houses and stores, and is to be found in thousands in the empty rain-water drains of an oriental town as well as in their badly laid sewers.

It will be noted that there is an essential difference in the habits of the two species of rats, the one living with the people in their houses, the other apart from them in the gullies and sewers, or in the open ground round the houses. There is a common meeting ground, however, for the two species in the ground floors of houses and stores, and as it is known that *M. decumanus* develops plague before *M. rattus*, and appears to be more severely affected by it, the importance of remembering this is obvious. It is plain from the above account of their habits that man is much more likely to take plague from *M. rattus*, with which he is brought into intimate relation, than from *M. decumanus*, which lives apart from him.

Some time after the Commission had begun work, Dr Hossack, who was on plague duty in Calcutta, published a paper calling attention to the fact that most of the rats caught in that city belonged to a third species of rodent, *Nesokia bengalensis* (Fig. 3).

It was then recognised by the Commission that a few of the rats caught in Bombay also belonged to this species, but as they amounted to 3.2 per cent. only of the decumanus-like rodents, they may for practical purposes be neglected. This small bandicoot rat may, like *M. decumanus*, be distinguished from *M. rattus* by

having the *tail shorter than head and body together*. The tail has well-marked rings like that of *M. rattus*, but it has not the brush of hairs projecting beyond the tip as *M. decumanus* has. The head is broader and shorter, and the ears larger than *M. decumanus*. The fur is dark and coarse, and numerous bristles are present on the back. Nesokia grunts like a pig when alarmed, whereas the other rats squeal.

In the course of his study of the rats of Bombay, Captain Liston likewise discovered that the fleas of rats belonged to a different species from those of man. He sent specimens to the Hon. N. Charles Rothschild, the greatest living authority on the Siphonaptera, who identified them as *Pulex cheopis*, a flea first found by him in Egypt (hence the name), but since ascertained to be the commonest rat flea in all parts of the world, with the exception of Northern and Central Europe, where it only occurs rarely on ship rats. It is the common flea of rats in India, and is found on all the rats there of whatever species. Liston likewise found that, in the absence of rats, this flea would take to other animals and even to man. Thus in March 1903, the guineapigs at the Zoological Gardens at Bombay were attacked by plague, and on investigating the matter, Liston found that the animals, and especially those that were sick, were infested with fleas, though, as a rule, none are to be found on them. Dead rats had been picked up near the guineapig cages, and so he concluded that the rat fleas, having no rats to feed on, had attacked the guineapigs, and thus infected them with plague. He therefore hit on the happy idea of using guineapigs for trapping fleas in plague-infected places, and in this way was able to prove their presence in huts from which plague patients had been removed.

On dissection, these fleas were found to have their stomachs full of plague germs in an apparently living and healthy condition, in no way adversely acted on by the juices of that organ. He likewise found that a certain proportion of the guineapigs exposed to the bites of these fleas developed plague and died of it. Guineapigs exposed in neighbouring huts which were not plague infected, had no fleas on them, nor did they die of plague. He likewise proved by actual observation that *P. cheopis* will attack man when its natural host is absent. For instance, he relates that rats began to die in a crowded tenement or "chawl," then they disappeared, and the inhabitants were so troubled with the attacks of fleas that they had to sleep in the verandahs. A few days later two of them developed plague, and on collecting fleas from the others, it was found that out of thirty caught, no less than fourteen were rat fleas.

These observations of Liston's seem to me to amount very nearly to incontrovertible proof that plague can be conveyed by fleas from one animal to another, and that the rat flea will attack man, and therefore may transfer it to him also.

Such was the state of our knowledge in India early in 1905, and so important to us in the laboratory did the flea hypothesis appear, when viewed from this new standpoint, that Captain Liston and I designed, and obtained permission from the Bombay Government, to build a set of experimental godowns or cabins, in which to carry on a series of experiments in circumstances as nearly approaching those found in the native town as possible. These godowns were complete before the Plague Commission arrived, and it was only because we had to await the necessary drying of the walls that experiments had not already been begun before they were handed over to that body. When the Commission's experiments are described it will be seen how important a part they played in the proof of the infectivity of the rat flea.

The godowns are six in number, and built as a row of " lean-tos " against an existing wall. The walls, of brick and mortar, are 9 inches thick, and the floors are of concrete on a high plinth. The floor space measures 7 by 6 feet, but an inspection chamber inside the door, measuring 3 feet square, has to be deducted, thus leaving only 33 square feet available for the experimental animals. The front walls measured $7\frac{1}{2}$ feet, and the back walls 9 feet in height. The roofs were lined inside, below the tiles, with wire-netting, so as to prevent the entry of wild rats through the tiles.

The inspection chambers also were shut off from the interior space where the animals were kept, by similar wire-netting, so that one could open the door and, standing in the chamber, watch what was going on inside, without the danger of letting any of the infected animals escape. The six godowns were in all the above respects exactly alike, except that Nos. 5 and 6 had windows in the walls to permit of ventilation, but closed by a double layer of wire-netting carried on a wooden framework. The essential difference is in the structure of the roofs.

Numbers 1 and 2 were covered with the ordinary country tiles, laid on wooden laths, such as one finds everywhere in India. The tiles are made by the potter in the shape of cylinders, which are divided lengthways before being fired. They are laid in four layers on the laths, the lowest layer with the half cylinder facing up, the second facing down so as to cover the edges of these ; and so again

with the two most superficial layers. It is evident that such an arrangement admits of easy dislodgment by rats and crows and is in no sense a rat-proof roof.

Godowns Nos. 3 and 4 were roofed with flat pan tiles, made in Mangalore, and known universally as "Mangalore tiles."

These do not admit rats so readily as the country tiles, but yet are by no means "rat-proof."

Godowns Nos. 5 and 6 were covered with corrugated iron sheeting, carefully cemented down to the top of the walls, to make these godowns rat-proof. As this did not, however, prove effective against fleas, a reinforced concrete roof was substituted.

The only other difference between the godowns was that No. 1 had a glass skylight, and No. 3 a ventilator, which admitted a certain amount of light to the interior, whereas the others were nearly dark. By this arrangement we were able to carry out experiments under various conditions, and it was found that the number of fleas in each godown varied with the accessibility of the roof to rats and the amount of light in the interior.

With all this evidence before them, the Plague Commission decided that rats were responsible for the spread of plague in India and therefore set themselves to find out how this took place.

Three possibilities presented themselves for their consideration, viz.:—

(*a*) Rats might soil the food by their excreta.

(*b*) Rats might similarly contaminate the floors.

(*c*) Some insect on the rats might convey the bacillus.

Let us see what conclusions they arrived at as to these three alternatives.

(*a*) That infected food might be the means by which plague is conveyed from one being to another, was a favourite hypothesis in the early days of plague, and gave rise to much vexatious legislation.

The first Plague Commission concluded that though it was possible to produce plague by feeding, yet this method was probably not a common one in nature. The German Plague Commission thought that rats generally became infected in this way in nature. The Austrian Plague Commission succeeded in infecting rats through the mouth, but never saw any *post-mortem* appearances pointing to primary infection through the stomach, unless a stomach tube was used, or a large amount of material was administered. Other observers on the Continent and elsewhere, including Klein in London,

have succeeded by various methods in infecting rats by feeding with cultures or the bodies of animals dead of plague.

It therefore became important to reinvestigate this question. The Commission accordingly undertook three series of experiments, using the ordinary wild rats of Bombay; rats from ships in the harbour; and rats found in the Punjab villages. These were fed in the same manner with the internal organs of rats or guineapigs dead from acute plague, mixed with the ordinary food of the rats. The Bombay rats were each fed once on from one-sixth to one-tenth of such internal organs, while the Punjab ones got slightly more. A fourth series of experiments was carried out to get over any objection that might be raised from the absence of bone in the food given to the first three sets of animals, by giving the carcases of dead plague rats to Bombay rats which had been starved for twenty-four hours. A rigorous *post-mortem* examination of all rats dying was of course made, and those left alive at the end of three weeks were killed and similarly examined.

The result is set forth in the following table :—

Series.	No.	No. dead of plague in 21 days.	No. plague-infected when killed, on 21st day.	Percentage plague-infected.
I. Bombay rats.	415	83	6	21.4
II. Ship rats. .	41	17	1	43.9
III. Punjab rats. .	28	19	0	67.9
IV. Bombay rats, fed on carcases.	108	36	5	38.0
Total . .	592	155	12	26.2

Note that the ship rats were twice as susceptible as the wild rats of Bombay city, also that the Punjab rats were three times as susceptible. This latter fact is probably to be explained by the larger dose of infected material given, for the rats also all died within five days of feeding, which implies a more virulent disease. From these experiments the Commission arrived at the conclusion that it was possible to infect rats by feeding, *if the dose of infecting material was large*, much larger in fact than would be likely to be met with in nature. That rats do not become thus infected in nature they considered proved by two considerations, viz., the different site of the primary bubo in naturally infected rats and in those infected by feeding as above, and the pathological changes to be found in the stomach and intestines.

In 6000 *post-mortem* examinations of rats naturally infected with plague they found the primary bubo to be situated in the neck in 74 per cent., and in the inguinal region in 25 per cent., of the animals, and in no single case were mesenteric buboes present. On the contrary, in the case of the 155 rats infected by feeding, the mesenteric glands were affected in 73 per cent., the neck glands in 35 per cent., and the inguinal glands in one case only.

Again in naturally infected rats the stomach and intestines show, as a rule, no marked pathological change, whereas in the case of those infected by copious feeding, well marked lesions are to be found there.

The large number of times that negative feeding experiments with grain soaked in plague culture have been made in India by Gibson and others (Pl. Com. Rep., vol. v. p. 74), proves that contamination of food stuffs, such as might conceivably occur in nature by dropping of plague-infected excreta, would not prove dangerous, but to confirm or disprove this, a number of investigations were made as to the infectivity of the urine and faeces of plague patients and rats.

Comparative examinations were made of the numbers of bacilli to be found in the blood and urine of rats dying of plague, by taking blood from the heart, and urine from the bladder simultaneously, and estimating the actual numbers of bacilli in a cubic centimetre of each.

In 21 cases where this was done, two showed bacilli neither in the blood or urine; in 13 no bacilli were found in the urine though present in the blood. In one instance where the blood contained from 10 to 100-million plague bacilli per c.c., less than 10 per c.c. were present in the urine, and in cases not so extreme as this the number in the urine was always from 100 to one million times less than in the blood.

To ascertain whether urine containing plague germs was a means of spreading plague among rats, a large number were fed with the urine of human plague cases, of so acute a nature that 88.5 per cent. of them terminated fatally.

Of the 35 cases, the urine of which was used, 10 had bacilli in the peripheral blood as ascertained by culture.

In all, 194 rats were fed once on such urine, from 2 to 3 c.c. being mixed with the parched maize on which the rats are ordinarily fed. Not one of them developed plague.

That the urine had live plague bacilli in it, is proved by the fact that guineapigs injected with it subcutaneously, died of plague in many instances, 7 out of the 13 urines being thus proved

infective. It is quite clear then that much greater contamination of food than could ever conceivably occur in nature would be required before plague could be spread in this manner.

As regards the chance of fæcal contamination of food giving rise to plague in rats, the following experiments were carried out. Rats in whose blood abundant plague bacilli were found, were used to furnish the material. After death the abdomen was opened, and with the usual minute precautions to avoid contamination of the fæces with blood or peritoneal fluid, the anterior wall of the rectum was divided, and a small portion of the contained fæcal matter removed. This was emulsified with a small quantity of sterile broth, and the thick emulsion resulting rubbed into the shaved and scarified surface of the abdomen of fresh rats. In this way, 139 rats were inoculated, but as of these 36 died of some other disease, only 103 were observed throughout. Of these only 3 died of plague, giving a mortality rate of 2.9 per cent. for this method of experiment. This method of inoculation by rubbing infective material on to the abraded abdomenal surface, is, I may explain, extensively used by us in the laboratory, for the diagnosis of plague in putrid carcases, where the microscope and culture methods fail. From extensive experiments made by the Commission to test the reliability of this method, they concluded that "it would appear to fail only in about 2 per cent. of fresh, and about 10 per cent. of putrid rats" (vol. vii. 358).

We may safely conclude that had it been possible for rats in natural conditions to be infected by the bacilli in the fæces, a larger number than 3 out of 103 would have developed the disease.

If, then, rats cannot be infected by the sure and effective method of skin inoculation, it is reasonable surely to argue that they would still less be likely to acquire plague by eating the excreta of their plague-stricken companions. And this is of course confirmed by the fact that one never finds mesenteric buboes in rats infected by plague in a natural way.

Again, as will be more fully explained later on, experiments on a large scale were carried out in a flea-proof godown, in which large numbers of guineapigs were confined, in circumstances of over-crowding and bad sanitation. The godowns were never cleaned out, so that at the end of a week the animals in them were running about on a floor which resembled nothing so much as a dunghill. Those who have watched guineapigs feeding, know how greedily they feed, not hesitating to climb into the feeding troughs, so that

the food soon becomes defiled with both solid and liquid excrement. Yet 150 guineapigs were kept under these conditions for six weeks at a time, and not one ever contracted plague, and this though at times there were introduced among them nearly their own number of infected ones. It is certain that the healthy animals must have eaten quantities of food defiled by their plague-stricken companions, yet as long as fleas were excluded none of them developed plague.

Again it has been a common observation in the Indian plague hospitals, that infants do not contract plague though suckled by plague-stricken mothers, and the same thing is seen among guineapigs in flea-proof godowns such as the above.

With regard to plague in India, we can say with perfect confidence in conclusion, that food contaminated with plague material has nothing whatever to do with the spread of the disease.

It may be well, however, to call attention to the observations of Hunter in Hongkong, who maintains that rats scatter broadcast, by means of their excreta, millions of plague bacilli, that they thus contaminate the food of the people, and that man is infected, in the majority of cases, by the intestines. He minimises the danger of skin infection, and considers that plague-infected fleas are of no importance, and that insects act merely as passive carriers of the bacilli. He considers buboes secondary to the blood infection. He is supported in these contentions by Simpson, who says: "The facility with which the lower animals contract plague by feeding is in favour of man contracting it often in the same way." It may be remarked here that though Simpson was able to infect all the domestic animals of China, with the exception of the dog, by feeding them on huge quantities of plague-infected viscera, yet the same experiments carried out by myself in Bombay, and by Hill and Pitchford in Natal, proved uniformly negative. Further, the Austrian Plague Commission, who devoted most of their time in Bombay to the investigation of the pathology of human plague, came to the conclusion that in an overwhelming majority of cases infection takes place through the skin; that a primary blood infection does not take place, as the bacilli are in the first instance taken up by the glands, and only secondarily invade the blood stream; that infection may also gain entrance through the mucous membrane of the mouth, nose, throat, the tonsils, and conjunctiva; and that in no single instance did anything point to a primary intestinal infection.

It would appear almost as though there were some curious

difference in plague in India and in China, for the above views seem incompatible otherwise.

(*b*) We may now turn to the second supposition and see whether there is ground for believing that plague is spread through contamination of the soil. This has been a favourite theory with many authorities, and one can easily understand this when the habits of the natives of India are considered. They go barefoot in most cases, even the wealthy taking off their shoes on entering the house. They likewise often eat their food from leaves placed on the floor, and it must, in consequence, often be exposed to contamination from the soil, even after it is cooked. From the time when Yersin described a bacillus recovered from the soil of a plague hut in China, which he considered an a-virulent form of the plague germ, many have tried to find it in similar situations, but in vain.

Thus in India, Hankin, Gibson, Leumann, Liston, and others have repeatedly examined soil without being able to find the plague bacillus. In one or two instances where it was isolated, the observers attributed their success to recent contamination of the floor with pneumonic sputum (*vide* "Journal of Hygiene," vol.vi. p. 199).

Gibson, in a series of experiments carried out in the Plague Research Laboratory, proved that rats injected with an emulsion of soil to which a minute quantity of plague bacilli had been added, almost invariably died of plague, but that if soil from an infected locality alone were used, they died of some other disease. Recent researches by cultural and inoculation methods, carried out at my suggestion in the same laboratory by Mackie and Winter, with artificially infected unsterilised floor-soil, show that plague bacilli rapidly perish in the presence of saprophytic organisms, and have completely disappeared after four days. The Plague Commission's experiments in this connection were made in a small godown, roofed with country tiles, and in every way similar to hundreds of dwelling-houses in Bombay. The floor was the usual one of rammed earth covered with a plastering of cowdung. A second series of experiments was made in a similar room in which the floor was of lime concrete. These floors were grossly contaminated by flooding them with a virulent broth culture of plague bacilli, and from these floors scrapings were taken at intervals of twenty-hours and rubbed into the scarified abdominal surfaces of rats and guineapigs. The contamination of the floors was of course very greatly in excess of anything that could conceivably take place in natural circumstances, and the manner of infecting the experimental animals

was likewise calculated to be much more likely to give them plague than a natural infection would be. Yet the Commission declare that a cowdung floor "can retain its infectivity for forty-eight hours but no longer, and that a chunam floor, treated in the same way, does not remain infective even for twenty-four hours."

Another series of experiments was made with animals penned on the contaminated floors and running freely about on them. Under these circumstances, which more nearly resemble the conditions in which human beings live, except in that the floors were so grossly contaminated, it was found that cowdung floors remained infective for twelve, but not for twenty-four hours after contamination, while the concrete ones kept their infectivity for six, but not for twelve hours. In these experiments only a very small percentage of the animals exposed to infection contracted the disease, and then only when the floors had actual pools of liquid on them when the animals were turned on.

Again, as we shall see, animals may contract plague when carefully protected from contact with the soil.

Lastly, we may refer to the fact, which will be dwelt on in a subsequent part of this paper, that just as many guineapigs contracted plague in disinfected rooms in Bombay city as in rooms not so treated. The disinfectant was a well-made acid solution of perchloride of mercury, of the strength of 1 in 750, which was abundantly proved to be capable of sterilising the soil of the rooms. Thus all chance of infection from germs in the floors was removed, and yet this did not prevent the animals running about in these rooms from contracting plague from another source. Needless to say there were present rat fleas, which are not inconvenienced by the perchloride of mercury.

It will be seen, then, that neither in contaminated food nor soil could the Plague Commission find any solution of the problem of the spread of plague in India.

(*c*) Let us turn now to the last possibility, viz., that some parasite of the rat might be the sought-for agent.

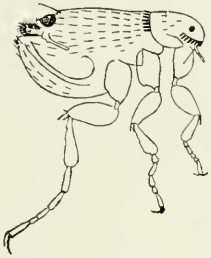
It soon became evident that the only biting insect that need be considered was the rat flea (*Pulex cheopis*), which, as we have seen, is the common rat flea all over the world, except in Northern and Central Europe.

As it is important to be able to distinguish one flea from another, the following simple description, taken from the Report of the Plague

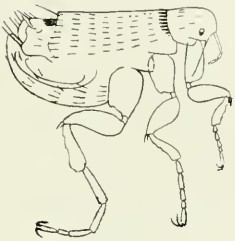
♂ Pulex cheopis

♂ Pulex irritans

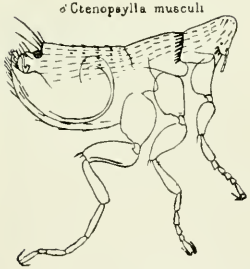
♂ Pulex felis



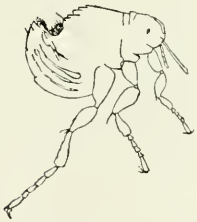
♂ Ceratophyllus fasciatus



♂ Ctenopsylla musculi



♂ Sarcopsylla gallinacea



Fleas commonly found on Rats in India. After Liston

PLATE IV.—TO ILLUSTRATE DR W. B. BANNERMAN'S ARTICLE.

Commission, is quoted as quite sufficient for the purpose; those wishing fuller information are referred to the paper itself.[1]

"These other species of flea are:—

"(1) *Pulex irritans*, the human flea.

"(2) *Ceratophyllus fasciatus*, the flea commonly found on rats in Europe.

"(3) *Pulex felis*, the cat and dog flea.

"(4) *Ctenopsylla musculi*, commonly found on mice and rats in various parts of the world.

"(5) *Sarcopsylla gallinacea*, a flea commonly found on birds.

"All the above-mentioned fleas have at one time or another been met with on rats. They all have well-developed eyes, except *Ctenopsylla musculi*. Of the remainder, *Ceratophyllus fasciatus* and *Pulex felis* have a prothoracic comb of bristles, and the latter a perioral comb. These bristles are absent in *Pulex cheopis*, *Pulex irritans*, and in *Sarcopsylla gallinacea*, but the latter flea is readily distinguished from the other two by its angular-shaped head, and its largely developed mandibles.

"We are thus left with *Pulex cheopis* and *Pulex irritans*, which can be differentiated from one another as follows:—

"(1) *Pulex cheopis* is small and light-coloured when compared with *Pulex irritans*.

"(2) The number of bristles on the head is greater in *Pulex cheopis* than in *Pulex irritans*, *i.e.* there is only one bristle on the posterior portion of the head in *Pulex irritans*, namely, the one which is found at the posterior and inferior angle of the head, and corresponds in situation to the bristle which is common to the two rows of bristles found in this posterior portion of the head of *P. cheopis*. Moreover, the ocular bristle in *P. cheopis* is situated nearly on a level with the upper border of the eye, while in *P. irritans* it arises nearer to the lower margin of the eye.

"(3) Theantipygidial bristles in *P. cheopis* are longer than those found in *P. irritans*.

"(4) In the female *P. cheopis* the antipygidial bristle is longer than the pyramidal bristle of the 9th tergite, while in *P. irritans* the pyramidal bristle of the 9th tergite is as long as, if not longer than, the antepygidial bristle.

[1] *Note.*—Since this paper was written, a new genus has been formed by Jordan and Rothschild, under the name Lœmopsylla (λοιμὸς = a plague, and ψύλλα = flea), to which *Pulex chiopis* is now referred. This flea will in future be known as *Lœmopsylla chiopis*. "Parasitology," vol i., No. 1, a supplement to the "Journal of Hygiene," March 1908; Cambridge University Press.

" (5) In the case of males the shape of the clasper at once distinguishes the fleas from one another.

" (6) The shape and size of the claws are different in the two species. They are small in *P. cheopis*, and large in *P. irritans*" (vol. vii. p. 455).

The first step taken was to find out whether it was possible to transmit plague from rat to rat by means of these fleas. The first set of successful experiments was made in the following manner: A glass box was made large enough to contain a couple of wire cages, in which rats or other small animals might be confined. Each cage was placed on a separate tin tray, filled with sand to absorb urine, and to afford shelter to the fleas, and was separated from the adjoining cage by a few inches of space. The tops of the cages were made of tin, and rose an inch or two above the sides of the glass box in which they stood, and had lids through which food and water could be introduced, and the animal withdrawn when necessary. The whole apparatus was covered and made insect-proof by fine muslin stretched from the sides of the glass box to the tin necks of the cages, and carefully secured with glue. The experimental animals in the wire cages were therefore protected from the visitation of insects from outside, and any insects placed in the glass box were unable to escape, though they had free access to the cages through the wire-netting of which the lower parts were made. Each cage being placed in a separate tray ensured that the excretions of one did not get into the cage of the other, in fact the experimental animals were living in absolute isolation, though in close proximity to one another, the only possible communication being through the air, or by means of some insect capable of passing from one cage to the other. The mode of procedure was as follows:

Into one of the cages was put a rat infected with plague artificially, and along with it ten to twenty rat fleas. As soon as this rat died a healthy one was placed in the other cage in the same glass box. The dead rat was left beside it in the first cage for from eight to twelve hours, when it was removed and examined to make sure it had died of septicæmic plague, for if no bacilli could be found in the blood, it is manifest that the fleas could not infect themselves by sucking. If the healthy rat died subsequently, the usual bacteriological examination was made to make sure it had died of plague. After the necessary practice had rendered the method workable, the experiment was made with white rats imported from England, and therefore not under suspicion of being partly immunised by

exposure to previous plague-infection. In eleven out of sixteen experiments, *i.e.* 69 per cent., the healthy rat contracted plague. The same procedure was then followed, using ordinary Bombay rats, which had been carefully segregated, to make sure they might not be in the incubation stage of plague. In fifty complete experiments it was found that in nineteen cases, *i.e.* 38 per cent., the healthy rat developed plague. Taking into account that a fairly large percentage of Bombay rats are immune to small doses of plague, these two results are very similar. We find then that on thirty occasions a healthy rat contracted plague from a plague-infected one in its vicinity in circumstances where the only means of communication was either " aerial contagia " or rat fleas.

To exclude the possibility of aerial infection, another series of trials was made by putting fleas from plague rats into a glass box, similar to the above, but containing only one cage with a healthy rat in it. In eight out of thirteen complete experiments made with English white rats the flea-bitten rat died of plague, a proportion of 61 per cent.

When Bombay rats were used, thirteen out of twenty-five contracted plague, a percentage of forty. To sum up, we find that in twenty-one out of thirty-eight trials healthy rats in flea-proof cages contracted plague in consequence of living with rat fleas collected from rats in another cage, dead or dying of septicæmic plague ; thus demonstrating directly that the rat flea is capable of carrying plague from one rat to another. Many hundreds of successful flea-transmission experiments were subsequently carried out with guineapigs in the above manner, and likewise by confining fleas from plague-infected rats in test-tubes, and allowing them to feed on definite areas of skin of healthy animals through a layer of muslin stretched over the mouth of the tube.

Having proved that it is an easy matter to infect susceptible animals by means of rat fleas taken from plague-infected animals, the next step was to find out whether close contact of one animal with another was, in the absence of fleas, capable of starting an epizootic among them. For this purpose the godowns already described were utilised. It is necessary to recall the fact that the essential difference in these six godowns or cabins consisted in the structure of the roofs, which gave more or less free entrance to, or excluded rats ; and that the number of fleas to be found in each godown varied with the accessibility of the roof to rats, and the amount of light in the interior.

The first experiment was made in godown No. 6, one of the flea-proof godowns, in the end of May 1905.

Fifty healthy and flea-free guineapigs, and ten others inoculated with a virulent culture of plague germs, were placed together in the godown, which purposely was never cleaned out, so that in a few days the interior became highly insanitary. The food, consisting of green grass and bran, was placed on the floor, and of course became contaminated by the excretions of the plague-infected animals. All the ten inoculated guineapigs died of plague with the exception of one which had been cutaneously inoculated, yet not one of the fifty healthy animals developed the disease, though they were kept under observation for seven weeks before being removed from the godown. Many other similar experiments were performed in the following years, both during the epidemic and non-epidemic seasons, and invariably with the same results, provided fleas were rigorously excluded. Thus in six contact experiments made in 1906-7, we find that "not a single healthy guineapig died of plague." In some of these experiments plague-infected guineapigs were added daily for from fourteen to twenty-one days, so that the intensity of the infection was well kept up. On one occasion it is recorded that "there were twenty-one plague-infected animals in contact with the twenty-five uninoculated guineapigs. These latter, however, remained healthy until the experiment was abandoned on Nov. 21, 1907. No fleas were observed on the guineapigs throughout the course of the experiment" (vol. vii. p. 426). A parallel series of experiments in the godowns which received a regular supply of fleas from the roofs was carried out at the same time. An example may be cited from one carried out in the epizootic season. On the 8th January 1907, five plague-infected guineapigs were placed in godown No. 2, and in three days they had all died of plague. On the 11th January, after the last of the dead bodies had been removed, twenty-five healthy guineapigs were introduced and allowed to run free in the godown. An epizootic of plague at once broke out amongst them, and all had succumbed in a fortnight. Fleas were present in great numbers throughout this experiment. Similar experiments made during the off season, resulted in the death of at most one or two guineapigs, and in many cases none of the healthy animals were affected at all.

At the end of one of these experiments an examination of the bodies of the last victims revealed the presence of innumerable fleas. Of these 115 were transferred to godown No. 5, where for three

weeks forty-nine guineapigs had been living in the insanitary conditions described above, yet without any ill effects. Next day 106 more fleas were added. In four days thereafter one guineapig was dead from plague, and in six weeks all had succumbed to that disease.

Still further experiments were performed in the flea-infested godown by separating the guineapigs in it into three groups: one allowed to run about on the floor, another confined in cages suspended two inches above the floor, and a third in cages two feet from the floor. It had been ascertained that the jump of a flea is less than six inches, therefore it is evident that the animals in the first two groups were accessible to the visits of fleas, while the animals in the cage hung two feet above the floor were not.

There were four guineapigs in each group. Two days after being placed in the godowns they were removed and examined for fleas under chloroform. "One hundred and twenty-five fleas were taken on the guineapigs" from the floor, twenty-seven from those in the cages hung two inches from the floor, and none on those in the higher cages. The eight animals composing the first two groups were evidently ill and had buboes on the neck. The four from the high cages were quite well. It only remains to say that all the animals in the first two groups died of plague, while the four in the cages beyond the reach of fleas remained healthy.

Monkeys, as more nearly resembling man, were used on several occasions in these experiments, being left in the godowns for forty-eight hours. "The result of these experiments was that not one of eleven animals protected from fleas contracted plague; while six out of thirteen unprotected developed the disease."

It will be convenient at this point to contrast in tabular form the positions of the buboes in rats infected by feeding, by fleas, and as found in nature, and we shall find remarkable support in this way for the theory that the rats found dead during an epizootic have contracted plague by being bitten by infected fleas.

	Naturally Infected.	Infected by Fleas.	Infected by Feeding.
Rats examined .	5,188	117	155
	Per cent.	Per cent.	Per cent.
Rats without buboes	16	18	8.0
Cervical bubo .	72	61	35.0
Axilary bubo .	23	22	0.7
Inguinal bubo .	18	35	0.7
Mesenteric bubo .	0	0	73.0

The remarkable similarity in the percentages of the first two sets of animals points undoubtedly to a common source and channel of infection, the source being the flea and the channel being the skin. While on the other hand the difference in these proportions produced by a new method of infection, and the appearance of buboes in a new site altogether, point as conclusively to this new method not being the one found in nature.

I cannot do better than sum up here in the words of Major Lamb, the Senior Member of the Commission, who writes (p. 41) —

"(*a*) Close and continuous contact of plague-infected animals with healthy animals, if fleas be excluded, does not give rise to an epizootic among the healthy animals. As the godowns were never cleaned out, close contact includes contact with fæces and urine of infected animals. Close contact, even the suckling of young by plague-infected mothers, does not give rise to the disease.

"(*b*) When fleas are present, the epizootic, if it does start, varies in severity and rate of progress according to the season of the year and the number of fleas present. The season in which epizootics are readily produced experimentally and spread rapidly, corresponds with that of the plague epidemic.

"(*c*) An epizootic of plague can occur in a godown containing infected fleas without direct contact of healthy animals and infected animals

"(*d*) In an infected godown the infection is effective in proportion as the test animals are accessible to fleas.

"(*e*) Infection can take place without any contact with contaminated soil.

"(*f*) Aerial infection is excluded.

"(*g*) The experiments lead to the conclusion that fleas and fleas alone were the transmitting agents of infection."

Having definitely shown that plague infection can be conveyed from one animal to another by means of the rat flea, experiments were made to prove that plague-infected houses are dangerous because of the fleas they contain. For this purpose houses were chosen, in which two or more cases of plague had occurred, or in which plague-infected rats had been found; that is, care was taken to be reasonably sure that the room experimented with was really plague-infected.

The method employed was that devised by Captain Liston, where guineapigs were allowed to run free in the rooms for from about eighteen to forty-eight hours. The guineapigs were then

caught and chloroformed in a bottle, when it was easy to collect and count the stupefied fleas. The first fact discovered in this way was that nearly all the fleas caught in plague-infected rooms were rat fleas, and that they were present in large numbers. The second fact discovered was that a large number of the guineapigs thus allowed to run free in these rooms developed plague. Thus out of a total of 189 houses experimented with in Bombay and the Punjab, forty, or 21 per cent., proved infective to guineapigs.

A similar set of experiments made in rooms which had been first disinfected by the Bombay Health Department is highly instructive.

The disinfection was performed by thoroughly washing floors and walls with an acid solution of perchloride of mercury of a strength of 1 in 750, which had been proved capable of killing plague-germs in cow-dung floors. A few rooms had likewise been treated with the fumes of sulphur, either alone or after the above. The thoroughness of these proceedings ensured the absence of plague germs from the earth and walls of the rooms; and yet out of thirty-one such houses it was found that guineapigs became infected in nine, a proportion of 29 per cent., or exactly the same percentage that proved infective out of forty-two other rooms experimented with in the same season, which had not been disinfected in any way. The average number of fleas caught in these disinfected rooms was forty, and in one instance as many as 263 were present. The average number in thirteen rooms not disinfected was thirty-four.

A second series of experiments was performed with fleas caught in plague-infected houses and brought to the laboratory in test-tubes, where they were placed in flea-proof cages with healthy rats or guineapigs.

In twelve experiments with fleas from rats, proved by examination to be plague-infected, there were nine successes (75 per cent.).

In ninety-six experiments with fleas taken similarly from guineapigs which had been allowed to run freely in plague-infected houses, there were twenty-six successes (27 per cent.).

"It is important to note that the great majority of the fleas caught on the guineapigs placed in the houses were rat-fleas (*Pulex cheopis*.)"

Yet another series of experiments in plague-infected rooms was carried out, with the object of showing that animals would contract plague, although protected from floor-infection and from all contact with plague-infected rats, provided they were not likewise protected

from fleas. For this purpose pairs of animals were placed in the rooms in cages, so contrived that one was flea-proof, and the other open to the entry of these insects.

Thus the floor and upper part of the cages was made of teak wood, but the lower portion of the sides was closed by a double layer of wire-netting, with a space of half an inch between the layers. This effectually prevented the animal inside from contact with the floor or with wild rats in the room, but did not exclude air or fleas. Half the number of cages were made flea-proof by substituting a layer of fine metallic gauze for the outer layer of netting. In each experiment a pair of animals, one protected by gauze and the other not, were placed in each room. They were left side by side for forty-eight hours, and then brought back to the laboratory, where fleas found on them were removed. In sixty-three experiments carried out in this way all the protected animals remained healthy, while eight, 13 per cent., of the unprotected ones developed plague.

A variation on the above experiment was made by protecting the caged animal, not by gauze, but by "Tanglefoot" fly-paper. The cages were modified so as to have a broad base, in the centre of which was the animal, surrounded by a six-inch area of this sticky paper, on which fleas would be caught when trying to reach the victim in the centre. A barrier of wire-netting was placed round the outer edge of this platform to keep off any wild rats that might enter the room. The control animal was in a similar cage, but the "Tanglefoot" was replaced by a layer of sand, so that the fleas might cross the intervening space. The result of fifty-nine such experiments was that all the "Tanglefoot" animals remained healthy, while 6 per cent. of those not so protected developed plague. The small number of successes in these two series of cage-experiments as compared with those in which the guineapigs were allowed to run about freely, is amply accounted for by the smaller number of fleas obtaining access to the animals.

Thus when the animals were allowed to run freely, the number of fleas per room caught was between twenty and forty, whereas in the case of the caged animals only an average of three fleas per house was taken.

Of the 401 fleas caught on the "Tanglefoot" papers, 221, or 55 per cent., were rat fleas, 148, or 37 per cent., were human fleas, and 32 or 8 per cent., were cat fleas. Plague bacilli were demonstrated in the stomach-contents of one out of eighty-five human fleas, and in twenty-six out of 132 rat fleas dissected.

As above noted, the most common site for the primary bubo was the neck, and it was there also that the fleas were commonly found among the fur.

If rooms are infective because thy contain rat fleas, one would expect to find more of them in plague-infected than in healthy dwellings. And this was found to be the case. In two years in succession observations having this end in view were made.

Thus in 1907 the houses observed were divided into three classes—

(*a*) Those deemed plague-infected, because a plague-infected rat was found in it, or a guineapig running loose in it developed plague, or plague-infected fleas were found in it, or fleas from it produced plague in a guineapig on which it was placed.

(*b*) Those presumably plague-infected, because a plague-case had occurred there, or because dead rats had been reported, though not actually verified by examination.

(*c*) Those definitely known not to be plague-infected, but which were situated in the neighbourhood of, and of the same type as plague-infected ones.

The following table shows at a glance the results :—

Class.	Total No. of Houses.	Total No. of Fleas taken.	Average No. of Fleas per House.
A	27	784	29.0
B	73	755	10.3
C	68	169	2.5

A glance at the third column of figures will show that the houses that were certainly plague-infected furnished nearly three times the average number of fleas that those only presumably infected did, and twelve times the number that uninfected houses did. Of 130 fleas that were dissected, 41, or 32 per cent., contained abundant plague bacilli.

To sum up we find :—

(1) Guineapigs may contract plague if allowed to run loose in plague-infected houses, and many rat fleas are found on them.

(2) In houses where all plague germs are killed by disinfectants, guineapigs still die in the same proportion, and an equal number of rat-fleas is found on them.

(3) Fleas caught on rats dead of plague are capable of infecting healthy rats or guineapigs not otherwise exposed to infection.

(4) Fleas from guineapigs which have been allowed to run loose

in plague-infected rooms, are similarly infective to healthy rats or guineapigs not otherwise exposed to infection.

(5) Guineapigs in cages which protect them from the floor or other source of infection, but which are exposed to the attacks of fleas in plague-infected rooms, contract plague in a certain proportion of cases. Those similarly placed, but protected from fleas, do not develop the disease.

(6) Most of the fleas caught in the above manner are rat fleas, and have plague bacilli in their stomachs in 10.7 per cent. of those dissected.

(7) Almost all the primary buboes in animals thus infected are in the neck, and the commonest place for fleas to be found is also in the fur round the neck.

(8) Plague-infected houses have twelve times the number of fleas in them that those free from suspicion of infection have.

The logical conclusion from the above premises would appear to be that rooms are dangerous because of the infected rat fleas in them, and that the soil and air have no infective properties.

Having proved that the means of transmission of plague from rat to rat is the rat flea, and that houses and rooms are infective only when they contain infected rat fleas, we must go a step further and consider what evidence exists to show that this same insect is the cause of the transmission of plague from rat to man, and that it is by its means that plague is spread from place to place.

In considering how man becomes infected it is important to remember that the avenue through which the germs gain access to the organism is the skin, in so far at least as the common bubonic and septicæmic forms of the disease are concerned. Pneumonic plague may be ignored, as it occurs in only about 2.5 per cent. of all cases.

It is well known also that the primary bubo develops in the glands in connection with the lymphatics draining the area of skin through which the germ enters. The first Plague Commission pointed out that the glands in various situations are affected in proportion to the extent of skin which they serve, and that there was therefore no "seat of election" for buboes. These conclusions are amply confirmed by experiments made by placing infective fleas on definite areas of skin in guineapigs, when it was found "that in practically every case the primary bubo developed in the group or groups of glands which stood in direct lymphatic connection with this area of skin." It has likewise been pointed out that rats

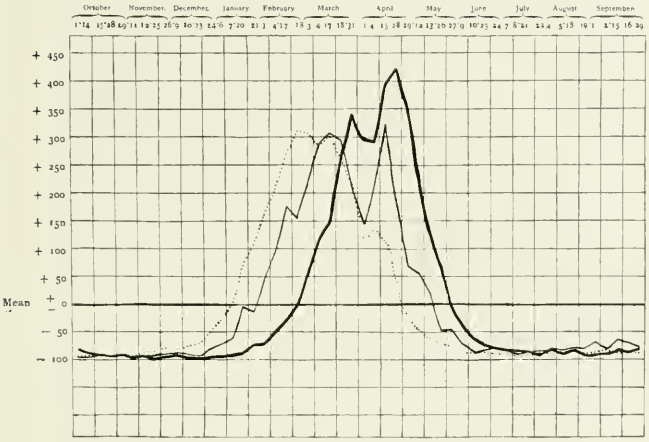


Chart showing the progress of plague in rats and man in Bombay City.

. . . . . Infected *M. decumanus.*

——— Infected *M. rattus.*

——— Plague deaths in man.

PLATE V.—TO ILLUSTRATE DR W. B. BANNERMAN'S ARTICLE.

wearing boots have just as great a proportion of groin-buboes as the barefooted Indians.

As has already been noted above, all evidence got in India, whether by the first Plague Commission, the Austrian Plague Commission, or the second Plague Commission, goes to negative the idea that plague is ever naturally conveyed through the digestive tract, and all evidence indicates the skin as the real point of entry. We have also discussed the evidence in favour of the actual agent being the rat flea.

If we study the chart showing the curves of rat-plague and human-plague deaths in Bombay city, we see clearly that there is a definite time-relationship between the *rattus* epizootic and the epidemic. Where the epidemic curve crosses the mean line we see that the interval separating it from that of *rattus* is about three and a half weeks; as it reaches the summit the interval is reduced to a fortnight. As the second apex is reached the interval is still further reduced to a week, so that the mean interval may be said to be from ten to fourteen days. Now, can this interval be accounted for on the flea hypothesis? I think it can. Thus, if following Major Lamb we divide the period into three, we find :—

(*a*) A period corresponding to that between the disappearance of the rat, the natural host of *P. cheopis*, and the time when the flea, pressed by hunger, will attack man. This was proved, by a large series of experiments carried out in the laboratory, to be three days. It was also found by experiment that fleas taken from plague-rats are most infective during the first four days, and then rapidly become less so.

(*b*) A period corresponding to the incubation stage of plague, which has been fixed by the first Plague Commission at three days.

(*c*) A period corresponding to the average duration of plague in human beings; this has been found to be about five and a half to six days.

If these three periods be added together, we find the period to be eleven and a half to twelve days, which corresponds very closely to what the facts of the epidemic in Bombay city require. That this is the true explanation is supported by certain observations carried out in the village of Kasel in the Punjab, as to the interval that separated the discovery of plague rats in a house and the subsequent appearance of plague among the inmates. "In five houses in which plague rats were found on only one occasion, prior to a human

case, the average interval between the two was ten days. In fourteen houses in which plague rats were found on more than one occasion prior to the human attack, the average interval between the finding of the first plague rat and the first case was twelve days, while the average interval between the last plague rat and the first case was seven days. These intervals correspond closely with those deduced from the curves representing the *rattus* epizootic and the epidemic both in Bombay and in Kasel itself" (Lamb, p. 51).

Further, we have already seen that plague-infected houses are infective to animals only because they harbour plague-infected fleas. We have likewise seen that monkeys are just as liable to be infected in the godowns as guineapigs, and they also contracted plague on several occasions when exposed in cages in plague-infected rooms. It would seem therefore not improbable that man exposed similarly to flea-bites from infective fleas would likewise contract the disease in this manner. Now Liston has shown that rat fleas attack man and other animals, not their natural hosts, in the absence of the latter. The Commission therefore made other experiments and observations to confirm this, and arrived at the following conclusions. When a man introduces his hand into a cage containing rat fleas, they will jump on to the skin, and if given time will bite. Unless very numerous, however, they will not attack the hand or arm in the presence of rats. When starved for seventy-two to ninety-six hours, fleas will readily feed on man.

When the attendant entered one of the experimental godowns in which rat fleas were numerous, he always brought out some on his bare legs, and it was the routine practice to station a man outside with a pledget of chloroformed cotton wool in his hand to capture these fleas. On one occasion when a servant went in four times in quick succession merely to remove cages forty four fleas were thus captured on his legs.

So common did the attacks of fleas become that the members of the Commission when visiting plague houses wore top-boots into which their trousers were tucked like so many Russian officers, or at least secured their trousers round their ankles by cycle clips. The bare-legged attendants who could not afford such luxuries were only saved from infection by regular injection of Haffkine's anti-plague vaccine. As an example one instance may be quoted. "In one house in particular, which the inhabitants had evacuated after a severe rat mortality, many rat fleas were caught on successive days on a man who went inside only for a short time, thus on April 18th,

51 ; on April 19th, 34 ; and on April 20th, 60 rat fleas were taken in this way " (Lamb, p. 54).

It is quite manifest, then, that in certain circumstances, and especially in the absence of rats, *P. cheopis* will attack man and feed upon him. Why should it not infect him likewise, as it does his near relation the monkey ?

Granting that the rat flea is the means by which man is infected, the question arises : How is it done ?

The Commission, by measurement and calculation, discovered that the stomach of a flea is large enough to contain the amount of blood necessary to enable it to imbibe many plague bacilli from the plague-infected rats of Bombay. They likewise found that the germs multiplied in the stomach in a large proportion of the fleas examined during the epidemic season, but in a much smaller proportion of those thus examined in the off-season.

After this multiplication, the germs are passed in abundance per anum and thus become deposited on the skin of the host while its blood is being sucked. There is no evidence to show that any transformation of the germ takes place in the body of the flea, nor that it can escape from it by the mouth or salivary duct. It has been experimentally proved that the fæces thus deposited are infective through the pricker wound, even without rubbing. The duration of infectivity of fleas has been estimated at fifteen days during the epidemic season and seven days in the off-season. Starved fleas died within a week, while those fed on human blood survive for three weeks. It is possible though not probable that a single flea could transmit the infection. Both male and female fleas can transmit the infection. *P. felis*, the common cat and dog flea, cannot transmit the infection, but both *Ceratophyllus fasciatus*, the common rat flea of Northern and Central Europe, and *P. irritans*, the species found on man, are capable of doing so, the former with ease, but the latter only with difficulty in the case of animals such as the guineapig at least.

All these facts have an important bearing on the question of the spread of plague from one place to another, which is the last point I have time to take up to-night. The first Plague Commission remark on this point : " In comparison with travellers, all other agencies which have been engaged in the distribution of plague from place to place are quite unimportant " (vol. v. p. 115).

The additional experience accumulated in India since this

opinion was given agrees with it and has led to the gradual withdrawal of restrictions on the importation of grain and other merchandise which an energetic Sanitary Department had in some cases recommended in the early days of plague.

If man, then is the sole means of introducing plague into fresh localities, how does he do it ?

We have seen that rat fleas will feed on man, but it was also discovered that though the fleas hopped on to the skin yet they often did not bite for some time. Also as fleas fed on man may remain alive for three weeks and may remain infective for fifteen days, there is ample time for fleas to be transported from place to place with passengers, and at the end of the journey be capable of infecting the rats of the new locality. It is a common experience that the man bringing plague to a village does not himself prove the first victim, he may even escape infection altogether. What happens is that the fleas brought by the visitor escape to the abundant rats in this plague-free village and infect them. This starts an epizootic, rats die and disappear, and the many infected fleas left behind attack man and produce an epidemic. Of course the importer may himself develop plague in the process, but this is not a certainty, many cases on record proving the contrary.

An importer even is not necessary, for clothes with fleas in them may be sent by post or otherwise, and their insect inhabitants prove infective up to a week at least That this may really take place is proved by experiments carried out with the clothing of plague patients. Such clothing placed in a flea-proof godown proved infective to healthy guineapigs, and on them were subsequently taken rat fleas with plague bacilli in their stomachs.

" In conclusion, then, we may state that all evidence points to the usual method of the importation of plague from an infected to an uninfected locality being by means of infected rat fleas and to the fleas being brought by human agency, namely, by healthy men, by plague cases, or in their belongings " (Lamb, p. 83).

The importance of the above researches in reference to the measures that should be adopted for the control of plague cannot be overestimated. They provide us for the first time with a scientific and sure basis from which to work, and have shown how futile were our efforts in the beginning, however well meaning they might have been. To set forth the bearing of these researches on the possibility or otherwise of separating the people from the rats, or of

exterminating rats or fleas would require another paper of at least equal length to this one. I would therefore in conclusion merely sum up in general terms.

GENERAL CONCLUSIONS.

1. Pneumonic plague is highly contagious, but being rare (less than 3 per cent. of all cases), plays a very small part in the spread of the disease.

2. Bubonic plague in man is not infectious, and is entirely dependent on the disease in the rat.

3. The infection is conveyed from rat to rat, and from rat to man, solely by means of the rat flea.

4. Plague is usually conveyed from place to place by rat fleas brought by people in their baggage or on their persons.

Note.—It seems necessary to explain why the name of Dr Ashburton Thompson of Sydney, who has been such a strenuous advocate of the flea-transference hypothesis, has been omitted from the above paper. It is simply because Liston had already arrived at his conclusions before Dr Thompson had done more than surmise that fleas were perhaps the cause of the plague in Australia. In Dr Thompson's own laboratory in Sydney, Tidswell tried experiments with fleas in 1900, but failed to confirm Simond's experiments, yet in spite of this the former inclined to the opinion that Simond was right.

This opinion he arrived at from the consideration of purely epidemiological facts collected by him during successive epidemics at Sydney. This first report was published in 1900, the second in 1903, and subsequent ones followed in each year up to 1906. His mature opinions will be found in a paper published by him in the *Journal of Hygiene*, vol. vi., for 1906. These reports are well worth reading as an example of what can be done by the collection and analysis of epidemiological facts alone, and they in every way confirm the conclusions arrived at by the second Indian Plague Commission, and are therefore of extreme value and interest.

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2. A CASE OF KALA-AZAR

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THE case we are bringing to your notice this evening presents many points of interest, but is particularly interesting for the reason that it is the first case of this disease which has been seen and recognised

in Edinburgh. It is not the first in Scotland, since another case which was seen by one of us ended fatally in Glasgow about two years ago. A melancholy interest attaches to the present case as the patient was a distinguished young graduate of this university who contracted the disease while engaged on research work in the Soudan.

Kala-azar may be defined as a specific disease, occurring in tropical and sub-tropical countries, characterised by fever of a markedly irregular type, enlargement of the liver and spleen, and progressive cachexia. The Leishman-Donovan body, the associated parasite, is found in the spleen and other organs and in the peripheral blood. The disease runs a somewhat chronic course and almost invariably ends in death.

Historical.—Though this disease has no doubt existed for ages, its true nature has only been worked out during the last five years.

About 1870, the disease, which at this time was regarded as a severe type of malaria, spread northward from lower Bengal along the Brahmaputra, and, assuming epidemic form, caused a huge mortality in Assam, seriously interfering with the cultivation of the land, whole villages being practically depopulated. In 1890 the Indian Government deputed Giles to investigate the matter; he came to the conclusion that the disease was a severe form of ankylostomiasis. This idea was not generally accepted. In 1896 Rogers was sent to Assam, and having found the malarial parasite in every case, a statement he was afterwards anxious to correct, reported that the disease was a malignant form of malaria.

Later, Bentley, who had long experience of the disease, showed that it was not at any rate malaria, as there was no periodicity. Quinine had no effect, and the malarial parasite was not usually present. He ventured the suggestion that it was a form of Malta fever.

Other theories, all equally vague, were put forward, but the true character of the disease was only recognised five years ago, the manner in which this recognition was brought about being most interesting.

In 1900 Leishman found in the spleen of a soldier who died at Netley of "Dum Dum" fever certain minute organisms the nature of which he was unable to determine; he put his specimens aside and did not pursue the matter further until 1903, when Trypanosomiasis having been recognised as a disease of man, he published a paper giving illustrations of the parasites, and suggested they were postmortem forms of degenerated trypanosomes.

Donovan in Madras had for some years previous to this been puzzled by numbers of cases coming to hospital suffering from fever with enlargement of the spleen. He recognised they were not malarial in origin, and had been in the habit of returning them as cases of enlargement of the spleen, cause unknown.

On reading Leishman's paper he performed splenic puncture in a number of these cases, and found the parasite on every occasion.

He published his results in the *Indian Medical Gazette*, and suggesting that the disease in Madras was probably identical with that prevalent in Assam, asked that those in charge of Kala-azar cases would by means of splenic puncture ascertain it if was so. Bentley at once wired that, acting on this suggestion, he had found the parasite in several cases, and thus the relation between the parasite and the disease was established.

Following this discovery the disease has been reported from India, Ceylon, Burma, China, Egypt, and many other places, and there is no doubt it is distributed all over the tropical and sub-tropical world.

It is now generally admitted that the disease is due to the Leishman-Donovan bodies, and many theories have been advanced regarding the mode of entrance to the human body. It is interesting to note that coitus, which has recently been claimed by Koch as one means of communication of Trypanosomiasis, was suggested by Rogers as a possible mode of infection in this disease—and various flies and biting insects were thought to be probable factors in spreading the disease. Recently Patton has proved that the bed bug not only acts as a carrier, but that the development of the parasite takes place in its stomach. Nicolle has within the last month reported cases in which the affection appears to have been conveyed to children by playing with infected dogs, and suggests that fleas may also convey the disease.

The parasite, which is usually found in enormous numbers in the spleen and also in the liver, bone-marrow, mesenteric glands, kidney, ulcers of the intestine and skin, and also, as shown by Patton, in most cases, in the leucocytes in the peripheral blood is a minute organism, $\frac{1}{3}$-$\frac{1}{4}$ the size of a red blood-corpuscle, presenting a very characteristic appearance. When lying free it is seen to be of an oval form, and presents two nuclei, one large and rounded, the other smaller and linear in shape, generally lying at right angles to the larger. Donovan has described a form resembling piroplasma occurring in red cells, but his observations have not been generally confirmed.

The true nature of the parasite has formed a fruitful source of discussion. They were classed by Donovan, Laveran, and Mesnil as piroplasmata, by Ross as sporozoa, and Leishman thought they were a stage in the development of Trypanosomes. Rogers, by a series of cultivation experiments, showed that they were young forms not of Trypanosomes, but of Herpetomonas, which differ from Trypanosomes in not possessing an undulatory membrane, and the flagellum comes off direct from the blepharoplast.

In our case attempts at cultivation were negative, probably owing to the small number of parasites present in the spleen, and the presence of numerous bacilli of the coli group, evidently a terminal infection.

The symptoms presented by this patient, to be later described in detail, were very characteristic of the disease; the daily double, and occasionally treble, intermission, as shown on the temperature chart, varied by periods in which the curve is most irregular, accompanied with but little distress, associated with a clean tongue, and no loss of appetite, the enlarged spleen, pronounced leucopenia; and the non-effect of quinine in large doses enabled us independently (having excluded malaria, enteric, paratyphoid, and Malta fever) to arrive at a correct diagnosis during life without resorting to splenic puncture.

One of the most interesting features of the case, is, that had we performed splenic puncture the result would probably have been misleading. After death it was found that the parasites were present in scanty numbers, and smears from certain portions of the spleen failed to show any parasites—had one of these portions been punctured during life the result would probably have been a mistake in diagnosis.

Patton has lately shown that if the blood is examined when the temperature is 102-103, it is generally possible to find some parasites in the leucocytes in the peripheral blood (repeated examinations of films in this case gave negative results), and it would appear that with this means of diagnosis at our disposal, together with the very characteristic features of the disease as a guide, splenic puncture, which has on numerous occasions been followed by fatal hæmorrhage, is not only inadvisable but unjustifiable.

The disease is not invariably fatal (Manson has recently reported a case of recovery in a European), but the mortality averages 96-98 per cent. of all cases.

We may now give the history of the case in detail.

Dr M. P., aged 25, left this country in September 1906 to carry out an exploring expedition with anthropological intentions in the Kurun district of the Southern Soudan. He passed through entirely unexplored country, and underwent very considerable hardships. He had various attacks of fever, which, of course, were not treated. He left Khartoum on the 17th of June 1907, at which time he was ill with fever. This was not unnaturally presumed to be malaria, but the blood was examined on one occasion at least and no parasites were found. He picked up somewhat on the voyage home, but felt seedy all the time; but on coming back to this country he again had constant fever with rigors occurring daily. He reached Edinburgh on the 18th of July, and on the following day sent for me (G. L. G.). I found him with a temperature of 105 at the culminating stage of an intense rigor, and he told me then that he had taken large quantities of quinine without any effect so far. I examined his blood, found a leucopenia, the leucocytes numbering only 4000, with a polymorph diminution, while the reds showed no very special change beyond a slight anæmia; no parasites could be found, either in films stained in the ordinary way, or with Ross's method and thick films. His general appearance, his furred tongue, the slight enlargement of the spleen, which could be felt under the edge of the ribs on deep inspiration, made me think it possible that he might have typhoid. His blood was Widalled and gave a positive reaction. As it was impossible to look after him at home, he was sent to the Fever Hospital here. I left for my summer holiday about the same time, and did not see him again till some time in September. During his stay there, it was, of course, ascertained that the disease from which he was suffering was not typhoid; repeated Widals were negative, as also the test for Malta fever, but its true nature was not suspected. He was treated in various ways, with quinine, with methylene blue subcutaneously, with arsenic, iron, etc., and the temperature, which at first was very irregular, with almost constant rigors, quieted down, and at one stage was normal for a day or so. He was very anxious to get up out of bed, and was allowed to do so. The temperature rose again, and practically never settled at all thereafter. One point of interest in the early stages was the slowness of the pulse. Though the temperature often rose to 105°, the pulse remained at 75 or 80. We have never seen this feature noted in the description of other cases. We have no note of the earlier blood counts while he was there, but on September 10th the reds were, 3,768,000; whites, 1250;

h.b. 60 per cent. On October 10th the count was, reds, 1,728,000 ; h.b. 30 ; whites, 938. This last is a sample of several counts made about the same time, which gave approximately the same figures. On my second visit, about the beginning of October, I had practically made up my mind that the disease was Kala-azar, and the same conclusion was come to independently by Major Marshall, who had seen the case several times. The spleen had enlarged very rapidly at one period. When this diagnosis was made, he was removed to Chalmers Hospital on October 22nd. His condition on arriving there was as follows : complexion pale and muddy coloured, but when the temperature was high, his face was often flushed and he looked temporarily better. There was considerable emaciation, and this advanced steadily as the disease progressed. The tongue was pale, rather dry, and tended to crack transversely. The digestion, at first poor, was under treatment very considerably improved, and up to the very end he was able to take his food with appetite and to digest it well. The bowels tended rather to be constipated, and aperients had to be given. There was a certain amount of distension of the abdomen at first. The liver was perhaps slightly enlarged, but the enlargement of the spleen and the slight distension of the abdomen made it difficult to appreciate this. The heart showed the usual systolic murmurs of anæmia, and was slightly dilated. The pulse ran from 100 to 120, and varied very much in force and character according to the temperature. The lungs showed no abnormality except a little pleurisy when he first was admitted. The urine was normal. There was no enlargement of the lymphatic glands, or of any of the ductless glands, but the spleen was very greatly enlarged, reaching an inch to the right of the umbilicus and from 2 to 3 inches below it, occupying thus the whole of the left side of the abdomen. On admission there was considerable tenderness from perisplenitis, which with a little counterirritation soon disappeared.

He steadily became weaker, though the anæmia did not greatly advance. The temperature was extremely irregular, rigors were frequent, and the range fluctuated between normal and 105°. But from the 27th October to the 9th November the fever was distinctly more of the continuous type. He became mentally confused, found it very weakening to talk, and slept badly. Soon after admission atoyxl was tried. A 10 per cent. solution was used, and of this 10 minims were injected daily at first ; this was increased on the fourth day to 15 minims, and on the eighth to 20 minims ; and this

was continued until he died. Absolutely no improvement resulted. The tongue became fissured, raw, and very painful, but he continued to take food well and to enjoy it. The bowels acted regularly until the 9th of November, when the temperature suddenly dropped to 90; the pulse became much feebler, and diarrhœa set in, the passage of fæces and urine being unconscious. The temperature thereafter rose, the diarrhœa continued, and he became mildly delirious, with hallucinations of vision. He died on the 13th of November, without further change. There had been no alteration in the physical examination of the organs for some time. The blood was examined very frequently; the red count varied comparatively little, running always about 2,000,000; the h.b. sank from 40 per cent. on admission, with a red count of 2,200,000, to 30 per cent. on the 5th November, with a red count of 2,000,000. The colour index was thus fairly high, though not actually up to 1, and the appearances in the films bore this out. There was, of course, marked poikilocytosis, and some polychromasia, and throughout a certain amount of granular degeneration in the reds, but no nucleated reds were ever seen. In the early part of the time he was under observation there was a fair number of large red cells. Towards the end the blood had more the character of a secondary anæmia, the red cells becoming smaller. The blood was examined in films several times after the last count was made, as he was too weak and irritable to endure the whole process. The leucocytes were 1750 on admission, and dropped steadily to 775. The proportions remained nearly normal, a typical instance being, polymorphs 60, lymphocytes 32, eosinophils 2. Neither I (G. L., G.), nor my resident physician, Dr Dykes, though we examined his blood at all periods, and both at the height and depth of temperature, ever succeeded in finding any organisms in the blood, nor were they found by Major Marshall, who also examined the blood with that in view.

The post mortem examination was made in the afternoon of the day when he died, with the following result. The spleen was greatly enlarged, and weighed 7½ lbs. There was some thickening of the peritoneal surface, with some adhesions to the diaphragm and to the abdominal wall. On section, nothing very definite was made out. The spleen was red, firm, but rather friable. There were a few infarcts here and there. The liver was also enlarged. It was pale and rather fatty. The heart was also fatty, with some dilatation, and there was a large amount of fluid in the pericardium, and some also in the peritoneum. In the stomach and small in-

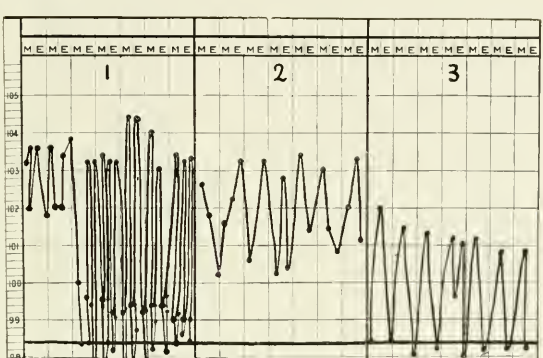

FIG. 1.—Temperature chart of three separate weeks. Showing extremely irregular character of the fever, two and sometimes three rises of temperature in one day.

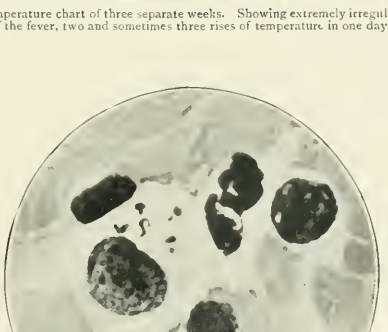

FIG. 2.—Smear from spleen. ×1000. Showing endothelial cell containing several typical Leishman-Donovan bodies.

PLATE VI.—TO ILLUSTRATE ARTICLE BY MAJOR MARSHALL AND DR GULLAND.

testine there was nothing to note. About the middle of the transverse colon an ulcer of about an inch and a half in diameter was found, with a somewhat thickened base, and a few smaller ulcers were found in other parts of the colon. The mesenteric glands were enlarged. The pre-vertebral hæmolymph glands were large and evident. The lungs, pancreas, kidneys, supra-renals, etc., showed nothing specially interesting. The rib-marrow was rather pale and fluid. Very careful research was of course made in the spleen, marrow, liver, the ulcer in the intestine, and elsewhere, for Leishman-Donovan bodies. They were only found, and then only in small numbers, in films made from the spleen, but were perfectly typical, as the accompanying illustration (Fig. 2) will show. Apparently they were distributed very irregularly through the spleen, as sections from various parts failed to show them. Dr Ritchie, of the Royal College of Physicians Laboratory, kindly made cultures for us from the spleen and liver. These were kept at a temperature of 22° Cent. in citrate-of-soda solution, but no growth resulted. This may have been due either to the fact that the portions of spleen used did not contain the bodies, or that there was a contamination by bacillus coli, which grew very rapidly, and possibly destroyed the bodies. In this case, therefore, splenic punctures as a means of diagnosis very probably would have given a negative result. The patient would not consent to it, as he knew the risk associated with it, and it was probably fortunate that it was not performed.

It will be seen from the comparison of the symptoms and the physical signs that this was a very typical case of Kala-azar from the clinical side, the essential point of difference from the Indian cases being its much shorter duration. The temperature was very characteristic, the double and sometimes treble daily intermittence in the early stages being very usual, as well as the frequent rigors, and the high continuous temperature with little distress of the later stages was also characteristic. The progressive anæmia, asthenia, and emaciation, the marked leucopenia, the constant absence of malarial parasites, the want of reaction to typhoid and Malta fever, were all as is usually found (Fig. 1). The occasional improvement followed by relapse, the great enlargement of spleen, the increase in size of the liver, the ultimate ulceration of the bowel, with serous effusion late in the disease, and the terminal diarrhœa, were all quite typical. The main point, of course, in which this case differs from the ordinary Indian cases was in the great difficulty with which Leishman-Donovan bodies were found in the spleen and other organs. This

feature of the case, the extremely small number and irregular distribution of the bodies in the spleen, appears to be characteristic of the disease as encountered in the Soudan; we have received a letter from a medical officer in Egypt who states, "Your case corresponds closely with some that have passed through my hands. I have found in three successive splenic punctures at intervals of about ten days, in the same patient, that the first puncture showed quite a fair sprinkling of parasites in three films, hardly any in the rest. The second puncture was absolutely negative, and the third only revealed two parasites in one film. Another case, films from which were sent to me from Khartoum, I reported to be negative after examining many films, but next day found a large 'mononuclear' containing three typical parasites and had to revise my opinion."

We were inclined at the time of death to attribute this absence to the very vigorous treatment with atoyxl which was carried out, but probably that had nothing to do with it, and we forbear to theorise without greater knowledge on the reason for this difference between the two forms. The appearances in the other organs, both naked eye and microscopically, are in accordance with what might be expected in any severe anæmia, and do not seem to be specially typical of Kala-azar. The case afforded exceptionally good opportunities for observation, as the patient was in hospital practically from beginning to end, and a two-hourly chart was kept throughout.

References.

1. Allbutt and Rolleston's "System of Medicine," vol. ii. pt. ii., "Kala-azar," article by Leishman.

2. Leishman. *British Medical Journal*, 1903, vol. i. p. 1252, and 1903, vol. ii. p. 1376; also 1904, vol. ii. p. 642.

3. Donovan. *British Medical Journal*, 1903, vol. ii. p. 79, and p. 1401; *Indian Medical Gazette*, 1903, vol. xxxviii. p. 478. *Lancet*, 1904, vol. ii. p. 744.

4. Nicolle. *Bulletin Pasteur Institute*, No. 9, May 5, 1908.

Meeting VII.—May 6, 1908

Dr James Ritchie, *President, in the Chair*

1. Election of Honorary Members

Douglas Argyll Robertson, M.D., LL.D., F.R.C.S.Ed., and J. Hughlings Jackson, M.D., LL.D., F.R.S., F.R.C.P.Lond., were elected Honorary Members of the Society.

II. Election of Corresponding Members

Ivan P. Pavloff, St Petersburg; Sir Henry R. Swanzy, M.A., M.B., F.R.C.S.I., Dublin; and Francis J. Shepherd, F.R.C.S. Ed., Montreal, were elected Corresponding Members of the Society.

III. Exhibition of Instruments

Dr T. D. Luke exhibited (*a*) a small piece of apparatus known as the "antivolat," for the gradual pouring of any extremely volatile liquid, which he had found valuable in the dosage of ethyl chloride. By means of it he showed that ethyl chloride could be slowly dropped and the usually explosive method of delivery entirely avoided. Messrs Reynolds and Branson of Leeds were the original makers.

(*b*) A new form of mask for the open drop method of etherisation now becoming so popular. This was one devised by Dr Ferguson of New Orange, near Boston, U.S.A., and possessed several advantages over Schimmelbusch's pattern. It consisted of a wire framework on which was clamped gauze in a thickness of 8-10 layers, and the whole mask then enclosed in a jacket of Canton flannel, in which a small aperture was left for pouring the ether on to the gauze. Dr Luke had found the mask very economical in ether, and efficient in the rapid induction of anæsthesia.

It was now made by Messrs Gardner, Edinburgh.

IV. Original Communications

1. A SIMPLE METHOD OF PREPARING CATGUT

By Professor John Chiene, C.B., M.D., LL.D., F.R.C.S.Ed.

When recently in Manchester, I saw in the operating theatre of Dr Archibald Donald a preparation of catgut which he had used for several years and found trustworthy, and which was prepared by his head nurse. I immediately asked the method, more especially as he claimed no originality, but could not tell me where he had learned the method. Its simplicity appealed to me.

Some years ago I had several cases which did not follow an aseptic course. I traced it to the catgut, not because it was badly prepared, but because the spool was taken out of the jar and put in again: it became contaminated in one of its many journeys before it was finished. It was therefore necessary to have the spools of small size, and the rule in my wards has since been, "Catgut once

taken out of the jar in which it is kept is never put back, any left over is thrown away." Each spool has rolled on it a metre of catgut, and there is very little loss.

At that time Mr Alexander of the Edinburgh Royal Infirmary, who has often been to me a most helpful friend, kindly prepared catgut for my daily use, which I have always since that time found reliable, but I also find that I have not been able to get my students when they become practitioners to use the method. I suppose it is too complicated, and certainly it requires a Jellett's steriliser. Dr Donald's simple method, therefore, appealed to me, and it will be best to give it in his own words.

"*Preparation of Catgut.* This is carried out by the house surgeon, or by the sister of my wards.

"The ordinary commercial catgut is thoroughly scrubbed on a board with soap and water and a brush. Then it is wound loosely round a wooden frame and soaked in ether for twenty-four hours. The skein is then re-wound on a long wooden frame, rather more tightly, but not so tightly as to prevent fluid from getting access to every part. The wooden frame with the skein is then put into a tall glass jar containing a solution of biniodide of mercury (one part) in absolute alcohol (five hundred parts), to which glycerine is added to the strength of five per cent. of the volume of the solution.

"It facilitates working if the lower end of the skein is divided and the longitudinal ligatures thus formed are kept in position by one or two cross ligatures. One can then get hold of a loop of catgut at the top of the skein by means of forceps,[1] and pull out a ligature of a definite length according to the length of the wooden frame), without removing the skein from the alcohol. I do not use the catgut until it has been in the solution for a month."

It was thoroughly tested in the laboratory, in the Surgical Department of the University, by Mr Mark Fraser, my house surgeon at the time, and Mr M'Murtrie, house surgeon in the Surgical Out-Patient Department, and was found to be sterile. I have tested it on the living subject, and in my opinion it is thoroughly reliable. It is well worthy of further trial.

Catgut by this method is now prepared by Mr Alexander, the only difference being that in the Edinburgh Infirmary the catgut is bought from the makers "fat free." The preliminary scrubbing

[1] I have got a special pair of forceps made by Mr Gardner, which does not injure the catgut on pulling it off the wooden frame.

with soap and water and the soaking in ether is not required, and all that the practitioner has to do is to soak this "fat free" catgut in the biniodide of mercury solution, which, I believe, was first introduced by Lockwood as a modification of the methods of Esmarch and Von Bergmann. There are now many ways of preparing catgut all tending to simplicity. Lord Lister has recently published the method of preparing chromic gut. It has its advantages, and for those who wish a dry catgut it is undoubtedly an excellent method. It must be soaked for a quarter of an hour in 1 to 20 carbolic acid before use.

A still simpler method is the aqueous iodine method of Claudius, recently brought under my notice. In it there is neither scrubbing of the catgut nor soaking of it in ether. A watery solution is made up, containing 1 per cent. each of iodine and of potassium iodide. The raw catgut is loosely wound on spools and placed in the solution. After eight days it may be used; indeed, it has been found that catgut, however infected, is invariably sterile within forty-eight hours. We are generally told that the catgut rots if left in this solution for more than a few weeks, and must therefore be transferred to an alcoholic solution of some sort. My informant believes that this is not true if the bottles are firmly closed with glass stoppers, so that the iodine cannot escape; he has used catgut which has lain for ten months in the aqueous iodine solution, without finding any reason for dissatisfaction. It may be slightly swollen, but is quite strong; still, there is no doubt that it is firmer, and threads more easily, if used within a few weeks of preparation. Raw catgut is preferable to the "fat free" variety for this iodine method. The latter is apt to be unequal in strength, strong at some parts, weaker at others, if anything is done to the gut in order to render the surface smooth. Macfarlan & Co. supply the Infirmary with gut which is only rendered "fat free."

2. COLOUR PHOTOGRAPHY IN THE TEACHING OF SURGERY AT LIVERPOOL

By Professor JOHN CHIENE, C.B., M.D., LL.D., F.R.C.S.Ed.

MR GEO. G. HAMILTON, F.R.C.S., Ed. and Eng., gave the students of the Edinburgh School a most interesting demonstration on Colour Photography by the Lumière process in the Anatomical Class Room in the University, in the end of March, under the auspices of Pro-

fessors Wylhe and Cunningham. Alexander Marshall, assistant in the Surgical Laboratory, has under my directions made a number of coloured photographs. Mr Hamilton used the lantern. I have never believed in a lantern in teaching, because there is only one man in the class who has seen the preparation the proper length of time; the other members of the class are divided into two groups, those who have seen the slide too long—the quick ones, and those who have not seen the slide long enough—the slow ones, in the class. Education must pass through the hands. The student must hold the preparation in his hand. This was the problem, and I show you to-night the solution, a small hand-mirror in a box. I have received much help from Mr M Kechnie, optician, 31 Castle Street, Edinburgh. The little box is made by Mr Alexander Macpherson 10 Forrest Road. I show also the same method applied to a stereoscopic coloured photograph. Let us bear in mind when we look through a stereoscope that John Goodsir was the first teacher to apply the stereoscope to the teaching art of medicine. This box will be shown at the Museum of the British Medical Association at the Sheffield meeting in July.

My great difficulty in teaching is to encourage students to use their eyes and their hands. Their ears are ever open to hear; would that they would see and handle! They would be better able by so doing to be reliable practitioners. The main work of teachers of surgery, which is in reality still a handicraft with brain cells as the motor, is to train students to see intelligently and to handle. Every student and most practitioners would do well to read and ponder over the truth as it is laid down in Dr John Brown's paper on Locke and Sydenham.

Sydenham said in leading his pupils into the wards of St Bartholomew's, "In entering this place, even this vast hospital, where there is many a significant, many a wonderful thing, you shall take me along with you and I will be your guide. But it is by your own eyes, and your own ears, and your own minds. *If Sydenham had been a surgeon he would also have said*, "*And your own hands*" and I may add by your own hearts that you must observe and learn and profit. I can only point to the objects and say little else than 'See here and see there'." A student who holds in his hand a preparation or a photograph is half way to understanding its true significance. Much has been done by the preparation of specimens so that they can be handled, and much will be done by handing to students coloured photographs instead of running them through on a lantern

screen. I have a friend who teaches from the *corner* of a square class-room. I asked, "Where do you hang your diagrams," He pointed to the lantern in front of him. Diagrams soak into a student; lantern slides glide past his mind like the fairies in "A Midsummer Night's Dream." "All that I have to say is to tell you that the lantern is the moon, I the man in the moon, this thorn bush my thorn bush, and this dog my dog" (Act V. Scene 1). Lanterns will have their day. They are communistic; and, as Le Bon says that the mind of a crowd lowers itself to the lower intelligences in the crowd —hence the follies and the cruelties which we owe to crowds—so a few good diagrams, and the handling of a stereoscopic coloured photograph à la Lumière, are intensely individualistic, and necessarily have an elevating influence on the minds of the average student—we must, if it be possible, lift him up, and draw out the Self in him.

3. SOME CONSIDERATIONS REGARDING PNEUMONIA IN CHILDREN DRAWN FROM AN ANALYSIS OF FIVE HUNDRED CASES

By G. H. Melville Dunlop, M.D., F.R.C.P.Ed.

That pneumonia in children is a very interesting, a very common, and a very fatal disease will, I venture to think, be generally admitted. During the period I have had charge of a ward in the Sick Children's Hospital, Edinburgh, 3300 cases of all varieties of disease have been treated in it, and of these over 500, or nearly one-sixth, have been cases of pneumonia, clearly proving, if proof were needed, how largely it bulks amongst the serious diseases of the children of the poor, necessitating treatment in hospital.

Much has been written on the subject, and it has been so thoroughly discussed in all its phases, and the field so carefully gleaned, that I fear my contribution will not add very materially to what is already common knowledge regarding the disease. Still, I consider that when one has had a large experience, it is desirable that that experience should be critically digested and summarised, and it is more with the object of ascertaining what I myself have learned, than with the view of contributing any new facts of material importance, that I have written this paper, and ventured to bring it before the Society to-night.

I was confronted at the threshold of my inquiry with the consideration of the method to be adopted in the grouping and classification of my cases—a question bristling with many difficulties and pitfalls, whether approached from a bacteriological, a clinical, or a pathological standpoint.

Broadly speaking, pneumonia in children may be divided into cases of broncho-pneumonia, and those of the lobar variety, but owing to the many anatomical and physiological differences in the respiratory system of the child, compared with the adult, the discrimination between the two conditions is frequently puzzling and sometimes impossible, and the difficulties assume wider proportions as one's knowledge of the subject increases.

Doubtless in the majority of cases the process is absolutely typical of one or other variety, but there are cases occupying a position on the borderland which are very difficult to place, and others again of a mixed variety which seem to defy classification, whether we investigate them clinically or pathologically. Clinically, we recognise a case as belonging to the lobar type of pneumonia by its abrupt onset, its lobar distribution, its continuous high temperature, and its termination by crisis, which is followed by a speedy resolution; while we characterise another as broncho pneumonia by its slower onset, frequently following bronchitis, by the marked remissions of temperature, terminating by lysis, and which after a prolonged course is followed by a protracted convalescence. Were all our cases marked by these broad and characteristic symptoms there would be no difficulty in discriminating between them; but pneumonia in children assumes so many varieties and has the habit of developing so many unexpected eccentricities that we are often sorely puzzled in which group to place individual atypical cases.

Thus in a considerable proportion of cases of broncho-pneumonia the symptoms commence abruptly with a sudden rise of temperature, which assumes a less remittent variety than the usual type, and runs a less prolonged course. Such cases are styled primary broncho pneumonias, and can generally be recognised by terminating by lysis (though by no means always so), and by being accompanied by general bronchitis.

Sometimes again we meet with cases of lobar pneumonia which are characterised by a wandering disposition, creeping slowly from one part to another of the lung, the crisis in consequence being indefinitely delayed and the temperature not infrequently falling by lysis. Indeed a certain remittance of temperature and termination

by lysis is not an unusual occurrence in young children. It is also far from uncommon to find lobar pneumonia accompanied by bronchial catarrh in the same or opposite lung.

In hospital practice again, when a case is first brought under notice late in the disease, when a large patch of pneumonia has developed, and when the early history, as is often the case, is indefinite, it is sometimes difficult to determine in which category to place it.

Turning from the clinical to the pathological aspects, we do not find the elucidation of the question any simpler. An ordinary lobar pneumonia is easily differentiated by its lobar distribution and more massive consolidation, consisting of an exudation of cells, chiefly leucocytes, in a fibrinous matrix, and a broncho-pneumonia by its more widely disseminated patches of consolidation, the bronchi and alveoli being chiefly affected, and the cavities blocked by an exudation consisting mainly of epithelial cells. Having made this simple statement of fact, we by no means dispose of the many discrepancies which the post-mortem table brings under our notice. For instance, how frequently do we find cases of broncho-pneumonia with a lobar distribution, brought about partly by the confluence of adjoining patches, partly by collapse of the lung, and partly by thickening of the connective tissue surrounding the bronchi and alveoli? It is moreover by no means uncommon to meet with a double lobar pneumonia, and with a broncho-pneumonia confined to a single lung. In my own experience I have occasionally seen on the post-mortem table, cases displaying a lobar pneumonia in one lung and a broncho-pneumonia on the opposite side, and on at least two occasions I have seen the super-addition of grey patches of broncho-pneumonia, appearing like a mosaic on a previously hepatised lung.

The difficulty of discriminating between the two varieties even extends to the microscopic examination, for we have it on the highest authority that, in a case of broncho-pneumonia, we may have, in addition to the proliferation of the epithelial cells, an exudation of fibrin similar to what occurs in lobar pneumonia. The general impression seems to prevail that in the age of the child we have a valuable aid to the classification of the disease, broncho-pneumonia being regarded as the pneumonia of infancy, and lobar pneumonia as that of older children; and though this is true in a general sense, it is far from being a correct interpretation of facts. The explanation of the special liability of young infants to catarrhal

pneumonia is probably to be found in the fact that the smaller bronchioles and alveoli, which are the most delicate parts of the lungs, are lined by numerous immature proliferating epithelial cells, and offer little resistance to infective bacteria. While readily admitting the frequency of broncho-pneumonia in young infants, and its uncommon occurrence in children over four years, except as a sequela of some infectious disease, I wish to emphasise the extreme frequency of lobar pneumonia in young infants. In the Edinburgh Sick Children's Hospital, where over a third of the cases admitted are under two years of age, I have treated a large number of infants, whose ages varied from a few weeks to two years, suffering from lobar pneumonia, and frequently had the diagnosis confirmed by post-mortem examination. In view of these many difficulties, I was tempted to adopt a convenient method of classification which would escape the necessity of specifying the type of pneumonia present, by simply dividing the cases into the two classes of primary and secondary pneumonias. I felt, however, that this method, however convenient, might imply that I accepted the view that primary broncho-pneumonia and lobar pneumonia were practically one, and the same disease, differing only in their form on account of the anatomical and physiological peculiarities of the child's pulmonary system. As I wish to disassociate myself entirely from this view, and as there are other disadvantages connected with this grouping, I have classified my cases under the usual headings of lobar pneumonias and broncho-pneumonias, dividing the latter again into the two classes of primary and secondary. Each case has been carefully and critically examined before being classified, and I have endeavoured, as far as possible, to eliminate every source of error. The tendency on the whole has been to place doubtful cases among the broncho-pneumonias. On analysing my cases according to this basis I find that 147 cases belong to the lobar variety. 85 of these cases occurred in boys and 62 in girls, which is, I think, about the usual proportion, viz. 3 boys to every 2 girls. An immense majority of cases occurred during the winter and spring months, the cases during these seasons numbering 101, compared with 46 during the summer and autumn, as shown in the accompanying table:

Winter,	74
Spring,	27
Summer,	27
Autumn,	19

A comparison of the different ages at which lobar pneumonia occurred gives strong support to the statement I have already made, that it is a disease of very frequent occurrence in young infants. Forty-five of my cases occurred in children under 2 years old, 12 of these being in infants under 1 year, the youngest case treated being only 3 weeks old. The annexed table gives the numbers treated at different ages.

45 cases under 2 years (12 being less than a year).
42 cases between 2 and 5 years.
60 cases between 5 and 12 years.

An examination of the portion of lung affected shows the very decided preference of the disease to affect the bases of the lungs, the pneumonia being confined to the bases in 88 out of 147 cases; and in nearly one-half the left base was affected. This is quite in conformity with the experience of others, though I can offer no reasonable explanation why it should be so. Next in order of frequency comes the right base, closely followed by the right apex, and after a long interval by the left apex. Both lungs were involved 8 times, but I make this statement with reservation in the absence of a post-mortem examination, as, owing to the readiness with which sounds are conducted in children from one side to the other, and owing to the difference of the percussion note according to the expansion of the lung, I believe that double pneumonia is oftener diagnosed than is perhaps always justifiable. The annexed table gives the portions of the lungs affected.

Left base	63 cases.
Right base	25 ,,
Right apex . . .	23 ,,
Left apex	11 ,,
Both lungs	8 ,,
Whole of one lung . .	10 ,,
Site not ascertained . .	7 ,,

I have divided the cases into those manifesting no physical signs, those with a late development of physical signs, and those in which they were well marked. I think it may be accepted as a fact that a small proportion of cases of pneumonia do occur which do not give evidence of its presence by physical signs; I believe that this proportion would be less than it is frequently stated to be, if our methods were more accurate and our examinations were more thorough and

frequent. Probably it is more often an expression of carelessness on the part of the physician than of non-existing physical signs in the lungs of the patient. I have occasionally, when on the point of concluding that no physical signs were present, found a patch of well-marked pneumonia in a spot which, I think, one often fails to examine, viz., high up in the axilla. Still I think, admitting all these qualifications, there remains a small residue of cases in which no physical signs can be detected. Seven of my cases have been classified under this heading. I believe that the explanation of the absence of physical signs is to be found, either in the fact that the pneumonia was of the abortive type which never went on to consolidation, or that it was of small dimension, or situated so deep in the substance of the lung that the sounds were altogether deadened. My impression is that the greater number of these cases are situated either at the apex or at the roots of the lungs. A late development of physical signs is of frequent occurrence. It is often not until the third, fourth, or fifth day that they become apparent. Seldom do we find them delayed till a later date, though I have seen them manifesting themselves for the first time as late as the seventh or eighth day. I remember one such case giving me considerable anxiety. From the general appearance and symptoms of the patient, I was practically certain it was a case of pneumonia, but waited patiently for the development of the physical signs to confirm the diagnosis. I had, however, to wait till the ninth day, the day after the crisis, for that confirmation, when my anxieties with regard to the case had already been put to rest by the fall of the temperature and the improvement in the general symptoms of the patient. Cases like the foregoing, are very embarrassing when one is being pressed for an opinion, and can only give a provisional diagnosis.

I shall not detain you by giving a detailed account of the physical signs in well-marked cases.

My experience leads me always to be very apprehensive when I hear distant, faint, or absent breathing, or breathing of an indeterminate, jerky character, accompanied frequently by an almost tympanitic percussion note. These physical signs are very typical of an early pneumonia, especially when accompanied by loud, harsh, exaggerated, puerile breathing on the opposite side. To those unfamiliar with the examination of the chest in children it often appears as if the latter was the affected side. The only other two remarks I desire to make with regard to the physical signs are that

the crepitant râle, so distinctive of the early stage of pneumonia in the adult, is more frequently absent than present in the child, and that the physical signs disappear with extreme rapidity. I have frequently been astonished to find scarcely any trace of pneumonia left, two or three days after the crisis, and in the majority of cases in the course of a week they have almost entirely disappeared, with the exception of a slight impairment of the percussion note.

The chief symptoms observed heralding the onset of pneumonia were vomiting, headache, convulsions, pain, shivering, and diarrhœa.

Vomiting was by far the most frequent symptom, and occurred in nearly two-thirds of the cases. Indeed, in my experience there is no other disease of childhood, with the exception perhaps of scarlet fever, which commences so frequently with vomiting as pneumonia. In some cases the vomiting was frequently repeated for a day or two after the onset. Pain is a common symptom, but it is much oftener referred to the abdomen than to the thorax. The combination of vomiting with pain in the abdomen, which may cause the abdominal walls to be kept rigid, leads to mistakes in diagnosis. On no fewer than seven occasions have cases been sent into my ward with the diagnosis of appendicitis, which subsequently proved to be cases of pneumonia.

Convulsions do not occur so frequently as an initial symptom as is generally supposed. They were noted as marking the onset of pneumonia twelve times, and were generally accompanied by headache, drowsiness, and apathy, being the symptoms associated with the condition known as cerebral pneumonia, and usually described as being more frequent when the pneumonia is situated at the apex.

My cases give only very slender support to this view, as the bases of the lungs and the apices were each affected six times when cerebral symptoms occurred. It appears to me probable that these symptoms occur when large areas of the lung are involved, when the temperature is uniformly high, or when the toxæmia is specially intense. Convulsions occurring at the commencement of the disease, and taking the place of the rigor in the adult, do not seriously affect the prognosis. Far otherwise is it when convulsions occur late in the disease. My cases go to prove that they almost invariably herald an early and fatal issue. Convulsions rarely occurred in children over two years old. In older children, especially in cases between five and eleven years, cerebral symptoms manifested themselves chiefly by delirium, sometimes of a wild character, and at other times of a muttering type, and chiefly

marked at night. Delirium was accompanied, as a rule, by muscular twitching, picking at the bed-clothes, tremor of the tongue, and often by diarrhœa. These symptoms were associated with a widespread disease and with a high temperature. In none of the 25 cases in which delirium occurred was the child under 3 years old, and in the great majority he was between 5 and 11 years. In only two of the cases, accompanied by this symptom, did death occur. Herpes occurred 16 times, or in about 11 per cent of the cases, but this percentage cannot be relied upon, as the symptom was probably not always noted when present. It does not seem to accompany pneumonia in infants, as in only two of the cases in which it occurred was the child under 6 years. In no case where it was present did a fatal result supervene. In most of the cases over 3 years old the temperature remained high until the crisis, which in the greatest number of cases occurred on the eighth day. The various days on which the crisis took place are recorded on the annexed table.

Crisis occurred on	the	second	day in	2	cases.
,,	,,	third	,,	3	,,
,,	,,	fourth	,,	6	,,
,,	,,	fifth	,,	10	,,
,,	,,	sixth	,,	15	,,
,,	,,	seventh	,,	16	,,
,,	,,	eighth	,,	22	,,
,,	,,	ninth	,,	14	,,
,,	,,	tenth	,,	10	,,
,,	,,	eleventh	,,	7	,,
,,	,,	twelfth	,,	3	,,
	After	the twelfth	,,	6	,,

Abortive cases generally terminated between the second and fourth day, while in the cases of the creeping or wandering variety the crisis was frequently delayed beyond the twelfth day. In the most prolonged case of the series the crisis did not occur until the twentieth day. In children under 3 years, the temperature is not so continuously high, and termination by crisis does not so frequently occur as in older children. In my 147 cases 24 terminated by lysis, which is, I think, a small proportion. The highest temperature recorded in any case was 107·5 F., but any temperature above 105 F., seems to add greatly to the gravity of the prognosis, and in the majority of my cases was followed by death.

The crisis was sometimes followed by prostration and perspiration, but in no case did dangerous collapse occur. A post crisis supervened in 25 per cent. of the cases, and was generally of ephemeral duration, subsiding in about twelve hours. When the post crisis continued for any length of time it generally indicated, either the spread of the pneumonia to another lobe, or the onset of empyema.

The rate of the pulse has not the significance it assumes in the adult. It generally runs from 130 to 150, and may rise to 160 or 170 without seemingly affecting the prognosis. A rate exceeding 180 is generally to be regarded as a grave condition. I wish to draw attention to a symptom not often referred to, viz., the extreme slowness and irregularity of the pulse sometimes present during convalescence, indicating the profound toxæmic influence of the pneumococcus on the cardiac muscle. On two occasions a pulse rate of 40 is noted, and it is quite a frequent occurrence to find it between 50 and 60 a minute, certainly a very low pulse rate for a child. I have never, however, met with any case of sudden heart failure during convalescence in a child, by no means an uncommon occurrence in the adult. Empyema is the commonest of all complications, and should always be suspected when the temperature rises again after the crisis. It seems as a rule to follow pretty closely upon the heels of the pneumonia, and I have found pus in the pleura within two days of the crisis. The pus is generally of a thick creamy consistence, and contains the pneumococcus in pure culture. It is not always such an easy matter to recognise an empyema as may be supposed, for the chest may be full of pus, and yet we may have loud tubular breathing over the effusion, so that we have often to depend upon the exploring needle for our diagnosis.

But even this method fails us at times. In one case in which I suspected the presence of pus, I passed a needle on nine different occasions, and in none of them was I successful in obtaining pus. As the child was rapidly getting weaker, I asked my surgical colleague to open the pleural cavity. As is his custom, when the child was placed on the operating table, he inserted a large needle in three or four different places, and getting no pus he asked me if he would proceed with the operation. So convinced did I feel of the presence of pus, that I asked him to proceed, but anxiously watched the progress of the operation. He removed a piece of rib, incised the pleura, but still no pus was forthcoming. On inserting his finger,

however, he felt and pulled out a thick piece of lymph several inches square, which was followed by a regular gush of pus, which put an end to my anxieties.

There seems to be a considerable amount of confusion and misconception regarding the prognosis of pneumonia in children, some authorities maintaining that the mortality is small, varying from 1 to 5 per cent., while others maintain that it is a very fatal disease, the mortality being 27 per cent.

These discrepancies have doubtless arisen by statistics being drawn on the one hand from hospitals that do not admit infants, and on the other from Foundling Hospitals where only children under two are treated. In the Edinburgh Sick Children's Hospital, where children of all ages are admitted, we are in a better position to form a just estimate of the true prognosis of the disease. Out of 147 cases, 15 have died, being a mortality of 10.2 per cent. Under two years, of the 45 cases treated, 12 died, giving a mortality of 26.6 per cent. Above two years, of the 102 cases treated 3 have died, giving a mortality of 2.9 per cent. My conclusions therefore are, that below two years the prognosis is grave, but above that age it is exceeding favourable.

Turning now from the consideration of the lobar to that of the broncho-pneumonias, I find that there were 355 cases of the latter variety, of which 120 were primary and 233 secondary. Two hundred and twenty of these cases occurred in children under two years, giving support to the popular belief that this is *par excellence* the pneumonia of early childhood. The sexes appear to be equally susceptible, 182 boys and 171 girls having been affected. As in lobar pneumonia, winter and spring claim the great majority of victims, 225 cases having occurred during these seasons, as against 128 during summer and autumn. Of the 120 cases of primary pneumonia, 92 occurred in children under two years, and 30 were under one year, the youngest case occurring in a baby sixteen days old. The symptoms of primary pneumonia bear a striking resemblance to those of the lobar variety, so much so that West and others regard them as one and the same disease. The pneumonia commences suddenly with high fever, accompanied as a rule by headache, vomiting, sometimes convulsions, quick breathing, pain in the side, cough and restlessness. Vomiting was in my experience nearly as frequent a symptom as in the lobar type, and convulsions, dulness, and apathy occurred in a not inconsiderable

number. In most of the cases the temperature at once assumed a high level, varying between 103° F. and 106° F., but was almost invariably characterised by morning remissions and evening exacerbations, the daily average fluctuations amounting to about 3° F. Although the morning remission was the rule, I have the records of quite a number of cases where the morning temperature regularly assumed a considerably higher level than the evening. The duration of these cases varies considerably, but an examination of my cases goes to prove that they last a much shorter time than the secondary cases of broncho-pneumonia and run a longer course than those of the lobar type. The average duration of the fever was eleven days, and in the cases that ended fatally death occurred on an average on the twelfth day. Relapses were much less frequent in the primary than in the secondary forms. The usual termination is by lysis, but in thirteen out of the 120 cases the temperature subsided by crisis. Such cases are difficult to differentiate from lobar pneumonia, but the disseminated character of the lesion of the lung, the marked remission of temperature, the pallor, the cyanosis, the greater amount of dyspnœa, and the distressing character of the cough are the symptoms chiefly to be relied upon. I need not detain you with an account of the physical signs in these cases, which in all respects resemble those present in the secondary pneumonias. Some of the cases of primary pneumonia, especially when they occur in very young infants, are exceedingly puzzling and difficult to diagnose. We find the temperature suddenly shooting up to 104° F. or 105° F., the child appears to be suffering from severe toxin poisoning, is cyanosed, prostrate, and his breathing is shallow and rapid. On examining the lungs we may hear only harsh puerile breathing with few if any accompaniments, and there may be no change in the percussion note. The infant is dull, listless, and apathetic, and appears to be in grave danger, when, after lasting one or two days, the temperature subsides, the alarming symptoms rapidly disappear, and in a day or so the child is again quite well. I have been in habit of regarding these cases as abortive cases of broncho-pneumonia, chiefly on account of the great cyanosis and distress of breathing which is so characteristic of this condition, and but for which they might equally be regarded as abortive cases of lobar pneumonia. I think it a pity that the old-fashioned title of acute congestion of the lungs, which so excellently describes this condition, is so seldom used nowadays. It seems to me that a sudden and a very intense congestion of the lining membrane of

the smaller bronchioles and air cells sufficiently explains the alarming symptoms which arise.

Two hundred and thirty-three, or nearly a half of the whole series, were cases of secondary pneumonias. The diseases to which these pneumonias were secondary are too numerous to capitulate, but the chief conditions which were followed by pneumonia were bronchitis, measles, rickets, whooping-cough, diarrhœa, and influenza, besides many other general constitutional diseases which have a debilitating effect upon the child's health and therefore predispose to an attack of broncho-pneumonia. An examination of the ages of these cases shows a marked contrast to those of the primary type: 102, or over 42 per cent. being over two years old. After suffering from one or other of the diseases specified for periods varying from a week to several months, the temperature suddenly runs up, the pulse respiration ratio becomes markedly altered, the child looks distressed, becomes cyanosed, the breathing more laboured and rapid, and the cough more frequent and troublesome. These symptoms are, I think, sufficient to warrant us in diagnosing broncho-pneumonia, without our being able to discover absolute signs of consolidation in the lung. Such cases are often described as capillary bronchitis, but I have never been able to appreciate the point where capillary bronchitis ends and broncho-pneumonia begins. Indeed, I go so far as to affirm that whenever we are able to diagnose capillary bronchitis, we can confidently state at the same time that the child is also suffering from broncho-pneumonia, as it is almost impossible to imagine the small bronchioles being affected without an implication at the same time of the alveoli, and I cannot recollect seeing post mortem a single case where the process had terminated in the smaller bronchi. The range of temperature in these cases is most typical, having during the whole course of the disease a remittent type, the daily fluctuations amounting to anything from 3° F. to 5° F. I was surprised on analysing my cases to find that, contrary to my general impressions, the average case seldom showed a higher temperature than 103° F. In only 96 cases of my series did it assume a higher range, the thermometer recording a temperature of 104° F. in 50 cases, 105° F. in 30 cases, 106° F. in 8 cases, and 107° F. in only 2 cases. In most of the cases of this series from time to time repeated remissions of temperature occurred, the temperature falling to normal and remaining so in some cases for a single day, and in others for several days, raising the hope that the process was at an end. In many of the cases four, five, or six remissions occurred, only to be followed by

fresh exacerbations, marking the spread of the pneumonia to hitherto unaffected lobules, an extension which could frequently be closely followed by physical examination.

The duration of these cases is exceedingly variable, varying in my series from 2 to 95 days, the temperature dropping to normal in the average case about the twenty-third day. When the case ended fatally the average day on which death occurred was the twenty-fourth day. Many of the cases, however, dragged on for many weeks, some of them for months, with an intermittent temperature interspersed with remissions, relapses, and exacerbations, the child growing daily weaker and more emaciated, creating the suspicion, nay, almost the certainty, that the case was tuberculous, when quite unexpectedly the temperature gradually fell to normal, and the child made an uninterrupted recovery. Another point to which I would direct attention is the fact that in not a few, especially in very young and debilitated infants, extensive pneumonias ran their course with scarcely any rise of temperature. In three atrophic infants the temperature actually remained sub-normal throughout the entire course of well-marked and extensive cases of pneumonia. The general appearance of a child with broncho-pneumonia is very typical, and contrasts with that of one suffering from lobar pneumonia. The child struck down with lobar pneumonia has generally flushed cheeks, quick, shallow, but seldom laboured breathing, and his cough is not as a rule a very distressing symptom. In marked contrast is the generally distressed aspect of the child suffering from broncho-pneumonia, the extreme prostration, the laboured breathing and dyspnœa, the dilatation of the alæ nasi, the cyanosis, and the incessant hacking cough, all of which are almost constant symptoms, and make a very characteristic clinical picture.

The pulse and respiration are always increased in frequency, the former varying as a rule from 140 to 180, and the latter from 40 to 80, and, as will be gathered from this statement, the pulse respiration ratio is greatly altered. The alteration of this ratio I regard as the most important sign of the onset of pneumonia.

I have made an analysis of the site of the lesion in the lung in these 353 cases of broncho-pneumonia, and find that both lungs were affected 182 times, or in considerably more than half of the cases. The right lung alone was affected 83 times, the left lung alone 72 times, and in 16 the site was not ascertained. The bases of the lungs were affected 5 or 6 times more frequently than the apices.

The only three remarks I shall make with regard to the physical signs are (1) that auscultation is a more reliable method of examination than percussion, as in many of the disseminated cases no percussion dulness can be ascertained. (2) That the pneumonic patches are frequently of such small size that every inch of the lung requires to be carefully auscultated to detect them. (3) That, unlike cases of lobar pneumonia, resolution as a rule takes place very much more slowly, and is often a very tedious, long-drawn-out process. One attack of broncho pneumonia seems to some extent to predispose to another, as there is a history of the child having suffered from one or more previous attacks in 40 of the cases.

My experience leads me to give a grave prognosis when I see the cyanosis deepening, the distress and restlessness of the child increasing, the cough ceasing, the pulse becoming faster and weaker, the respiration shallower, and the child becoming drowsy and lethargic. Death is generally due to one or other of the following causes: (1) increasing exhaustion, (2) suffocation and toxæmia, owing to the bronchi becoming choked, and (3) to heart failure.

I wish to emphasise three points which have struck me in connection with cases having a fatal termination. (1) the large number of cases which die very suddenly of heart failure - this occurred in twenty-three of my cases. (2) the tendency of the temperature to mount higher as death approaches. (3) the great liability of the child to die during an attack of general convulsions. Ten cases terminated in this manner.

The prognosis of broncho-pneumonia varies greatly according to the age and condition of the child, the amount of lung involved, and the nature of the infection, but the prognosis in any individual case is always an uncertain quantity. It is perhaps the disease of childhood which best justifies the expression that "while there is life there is hope," as the most unlikely cases, after lasting for months, and giving rise to the gloomiest forebodings, often unexpectedly clear up, but on the other hand cases which appear to justify a hopeful prognosis, sometimes die suddenly and quite unexpectedly of heart failure. The death-rate is always a heavy one. Thus Keating gives the general death-rate at 49 per cent. and under 5 years at 75 per cent. Holt states the mortality of all cases as 65 per cent. Morse puts it at 75 per cent. Pepper at from 30 to 50 per cent., and Donkin at about 50 per cent. I am glad to say that an examination of my cases shows a very much smaller death-rate than those quoted. Out of 553 cases, 80 died, giving a mortality

of 25.1 per cent., and if we exclude 15 cases which died within 24 hours of admission, the death-rate is reduced to 20.9 per cent.

An analysis of all the cases of broncho-pneumonia treated in the Sick Children's Hospital during the last ten years gives a mortality of 28 per cent.

I find that the death-rate was considerably less among the primary than among the secondary cases. The following are the chief points to be considered in estimating the prognosis :—

1. The primary disease : those cases which occurred after measles, whooping-cough, and diphtheria were exceedingly fatal, giving a death-rate of 54.4 per cent.

2. The younger the child the worse the prognosis : 64 per cent. of the deaths occurred in children under 2 years.

3. The greater the extent of the lungs involved, the less is the chance of recovery : in 73 per cent. of deaths both lungs were involved.

4. The poorer the previous health, the graver the prognosis.

5. When the child suffers from severe rickets the prognosis is always grave : in 34 per cent. of the deaths the report states that severe rickets existed.

6. Diarrhœa seriously affects the prognosis : this condition was present in 24 per cent. of the deaths.

7. A temperature 105° F. adds greatly to the gravity of the cases : 60 per cent. of such cases died.

8. A pulse rate of 180, and a pulse respiration ratio of 1 to 2, or 1 to 1½, is always serious.

9. The highest mortality was in cases which had lasted several weeks.

I know of no disease of childhood where treatment, judiciously carried out, gives better results than in broncho-pneumonia. By avoiding on the one hand unnecessary interference with nature's efforts to bring about a natural cure, and by stepping in and giving judicious assistance when these natural efforts require help and stimulation, very much can be done to tide the child safely through the many dangers and sudden emergencies to which he is liable.

The chief aim should be to put the child into the best position to withstand the disease, and the indications for treatment are therefore (1) to prevent the digestive system becoming deranged ; (2) to do all in our power to maintain the strength and vitality of the

patient (3) to keep the action of the heart constantly under observation, and (4) to prevent if possible the spread of the inflammation to fresh portions of lung.

A supply of pure air is one of the most important considerations as one of the main causes of death is carbonic acid poisoning. All my cases have been treated in a large airy ward, kept at a uniform temperature, with abundant cubic space and cross ventilation. I consider the system of treating these cases in the open-air undesirable, as I cannot believe that the inhalation of a cold, raw damp air can act beneficially upon an acutely inflamed mucous membrane. The feeding was carefully attended to, and the bowels regulated so as to try and avoid diarrhœa and flatulent distension, which latter adds so greatly to respiratory embarrassment. The diet consisted chiefly of milk modified to suit the age and requirements of the child, chicken and mutton broths, and, if the child could digest them, simple milk puddings. Frequent drinks of cold water were always allowed. The clothing consisted of a Gamgee tissue jacket and a loose flannel nightdress, so as to permit of free and unimpeded breathing. My practice has been to surround the upper part of the crib with a tent, open in front, so as to permit the free access of fresh air. Inside the tent are hung towels wrung out of a solution of one part of eucalyptus oil to five parts of water. I am persuaded that the evaporation of the moisture and the volatilisation of the oil causes very considerable diminution of the cough, and it is possible that its antiseptic action tends to prevent the spread of the pneumonia to fresh portions of lung. I give the nurse instructions to frequently change the position of the child and from time to time to take him out of his crib and carry him up and down the ward, so as to lessen the risk of hypostatic congestion. I am a believer in the efficacy of lightly made jacket poultices made of one part mustard and five parts linseed meal. They are applied four or five times a day for a few minutes. They redden the surface, act as a stimulant, relieve pain, and appear to be comforting to the child. I generally continue these applications for a week or ten days or longer at the commencement of the pneumonia. In spite of much that has been said and written to the contrary, I consider that alcohol is the sheet-anchor upon which we have principally to depend in the treatment of broncho-pneumonia. I am of opinion that the great majority of cases require alcoholic stimulation. As a rule it is unwise to commence giving alcohol early in the disease, but it should be reserved until the symptoms indicating prostration or lowering of the vital powers manifest themselves.

When such symptoms arise alcohol frequently acts like a charm, strengthening and steadying the pulse, lessening the cyanosis, reducing the temperature, increasing the vitality, and most valuable of all procuring a much needed sleep. A few years ago, on the advice of a friend whose opinion I valued highly, I discontinued for some months the administration of alcohol, an experiment I have never ceased to regret, as my results were not nearly so satisfactory during that period. I give it in doses of 15, 30, or 40 minims, according to the age of the child, every two or four hours ; and once its administration is commenced, I generally find it necessary to continue it until convalescence is established. In all cases I give strychnine, in minim doses of the liquor, every four hours, as a routine practice. It acts as a cardiac and respiratory stimulant, as a stomachic tonic, and causes contraction of the muscular walls of the smaller bronchi, and expulsion of the mucus contained in them. The only cases in which I omit strychnine are when diarrhœa occurs, or when there is marked respiratory stridor. When signs of advancing cardiac strain make their appearance, evidenced by irregularity of the pulse and increasing cyanosis, I add to the strychnine minim doses of the 1885 tincture of strophanthus, finding by experience that strychnine and strophanthus better sustain the blood pressure than digitalis and have no deleterious effect on the stomach. I am a believer in the efficacy of the inhalation of oxygen, and have frequently seen improvement in the child's condition speedily follow its administration, the breathing and colour improving, and the cyanosis becoming much less marked. I regard it as a valuable remedy. The only means I advocate for the reduction of high temperature is tepid sponging and the cold pack. When sudden collapse of portions of the lung set in I recommend free stimulation with alcohol, and placing the child in a mustard bath. I have entirely given up the administration of cough mixtures, having seen little good result from them, and I consider that they are very liable to upset the child's digestion, which it is so important to keep in good working order. When the bronchi are choked with secretion, an emetic of ipecacuanha often gives relief, and, failing this method, I have sometimes seen good results from the administration of belladonna.

During convalescence, nourishing food, small quantities of good port wine, quinine, cod-liver oil, and change of air are the methods of treatment on which I place reliance.

I conclude this paper as I commenced, by remarking that though I have not wandered very far from the beaten track, trodden by the

teet of many others, I have endeavoured to state my own impressions, without referring to the work of others, and to give the results of my own experience for what they are worth.

Meeting VIII.—May 20, 1908

DR JAMES RITCHIE, *President*, in the Chair

This was a Clinical Meeting held in the Royal Infirmary, the special subject being Dermatology.

I. EXHIBITION OF PATIENTS

1. *Dr F. Gardiner* exhibited the following cases:

(*a*) A girl with an eruption typical of PITYRIASIS ROSEA which had come out suddenly a fortnight previously. There was no herald patch, and the limbs were affected, which is exceptional. Although there was very little seborrhœa of the scalp and no history of former seborrhœa, still the fact that the flexures of the axillæ and elbows, as well as the skin behind the ears, were affected, and that the patient was about the age of puberty, inclined him to the diagnosis of seborrhœa.

(*b*) A case of ERYTHEMA MULTIFORME, affecting the arms, hands, and feet in a married woman who two months previously had developed a hard vulvar chancre. One month ago she was examined by a gynæcologist who diagnosed syphilis, and prescribed mercury, this eruption appearing almost immediately after. The mercury had not been used for the last fortnight, but in spite of this the eruption had continued as bad as before. This fact excluded a drug eruption, and the condition being itchy, syphilis was probably eliminated. Since this it has yielded to the administration of salicin internally.

(*c*) A case of ERYTHEMA SCARLATINIFORME very like a drug rash, and involving the whole body. The patient had taken copaiba, but had stopped doing so for a month, so that it could scarcely be considered causative. Probably it was a toxæmic eruption, and in any case it had disappeared under the use of boro-glaucin ointment.

(*d*) A child who had been X rayed two years ago for tinea capitis, showing a splendid head of hair, but unfortunately a fresh attack of the same disease.

(*e*) A mother with ATOPIC ECZEMA and her child with tinea

capit, interesting from the point of view of those who considered that the two diseases had a similar causation.

(*f*) A case of FAVUS, showing no scutula, but broken hairs and scaling very like tinea capitis. The diagnosis was confirmed microscopically.

(*g*) Four cases of RODENT ULCER under X-ray treatment.

2. *Dr Cranston Low* exhibited—

(*a*) A case of PEMPHIGUS FOLIACEUS in a woman aged 53, on whom the eruption had begun eleven years ago on the scalp. At first the eruption was typical of dermatitis herpetiformis. At that time she was under Dr Allan Jamieson's care, and later in the Longmore Hospital. About three years ago the character of the eruption changed, the whole skin becoming more or less red and scaly. On admission to the wards under Dr Norman Walker's care, she was covered all over with a moist, oozing, exfoliating eruption. From time to time fresh crops of blebs have appeared, the appearance of the eruption being preceded by itching. The temperature was not above normal, and the general nutrition is good. She was treated by arsenic internally and also had seven injections (each of from 250-500 million staphylocci) of a vaccine made from cultures obtained from her own blebs. The improvement since her admission is very marked indeed.

(*b*) Case of early MYCOSIS FUNGOIDES in a woman aged 52. The eruption began fourteen months ago in a condition which looked like psoriasis of the palms and soles. The eruption gradually spread up the limbs and eventually affected more or less the whole body with intervals of healthy skin here and there. Like the preceding case, she is being treated in the wards under Dr Norman Walker's charge. Some of the areas looked somewhat like lichen planus, others were more like pityriasis rubra. Sections cut from a piece of skin excised from the leg last December show some thickening of the malpighian layer, and cellular infiltration around the vessels of the corium. The section is not characteristic of any disease, but does not present the features of psoriasis. About a month ago on the backs of the feet and toes lumpy thickenings of the skin were noticed. These have slowly increased, and are raised slightly above the level of the skin. The condition is probably one of mycosis fungoides, the first eruption, in its indefiniteness, being quite characteristic of the pre-tumour stage of the disease. The feet are being treated by X-rays.

(*c*) A case of KERION of the scalp on a boy. The patient shows

two large spots on the scalp. The peculiarity of this case is that it is due to the small spore fungus of ringworm. Out of other 6 cases of kerion seen in the last 14 months, 4 were due to the small spored fungus.

3. *Dr Cranston Low* exhibited for *Mr Miles*.—A case of RUPIA SYPHILITICA. The patient was a married woman aged 21, a patient in the Lock Ward, who had first shown symptoms in December last. In November last, her husband was treated at the Male Lock for a primary sore. The eruption began on her legs, and spread upwards. It affects the whole trunk, face, head, and limbs. It shows the typical limpet like crusts. The lesions in places are circinate in arrangement. There is superficial ulceration and scar formation. On the lower limbs there are deeper ulcerations covered with crusts (ecthyma syphilitica).

4. *Dr J. S. Fowler* exhibited.—A case of URTICARIA PIGMENTOSA. The patient was a child aged 15 months, and the rash had been present ever since he was six weeks old. It was uniformly distributed over the trunk and limbs, and consisted of yellowish brown livid papules about the size of a large pea, closely set, and tending to be arranged along the natural lines of the skin. Superficially it bore a certain resemblance to a papular syphilide. Wherever the skin was pressed upon or irritated in any way, the spots took on an urticarial, wheal-like appearance. Factitious urticaria could be produced by stroking the skin with a blunt instrument. The case was perfectly typical in regard to the date of onset (urticaria pigmentosa being a disease which always begins in infancy), (b) the turgescence of the spots produced by pressure, and (c) the occurrence of factitious urticaria.

II. EXHIBITION OF SPECIMENS.

Mr Henry Wade exhibited—

(a) Two specimens illustrative of the condition known as HYPERKERATOSIS PLANTARIS. They had been obtained post mortem from a patient who had been an inmate of a lunatic asylum. The condition had been present for many years, and had caused no inconvenience when walking, although both feet were studded with numerous large growths like cutaneous horns.

(b) Foot amputated for MYCETOMA. The specimen was obtained from Hyderabad, Sind.

III. Original Communication

A REVIEW OF SIXTEEN MONTHS' WORK IN THE SKIN DEPARTMENT OF THE ROYAL INFIRMARY, EDINBURGH

By Norman Walker, M.D., F.R.C.P.Ed., Physician in Charge

The fact that after prolonged wanderings the Skin Department has at length found a home which, as an Irishman might say, is for a time, at least, permanent, has given me greater advantages than were available to my predecessor. Of these I have endeavoured to make use, and to-day I present some of the results of my sixteen months' experience. After three months of experiment we adopted the card system as the most efficient means of keeping the cases; the numbers are so great that books were out of the question. Certain special cases are kept on large cards, on which can be stamped outlines on which the distribution of any eruption can be graphically shown. The system requires that everyone concerned shall do his or her part thoroughly, and I think I may claim that considering we have in those sixteen months seen and taken notes of nearly 2500 cases, and that we can lay hands in a moment on a record of each, testifies to the care shown by my assistant physicians, the house physicians, the nurses, and my lady secretary, who is in ultimate charge of the cards. From the first set of cards she makes a cross index of diseases, so that I can at any time lay hands on any case or group of cases required to illustrate a lecture. Once a month the cases are tabulated on a half sheet of paper, and we thus have a very ready means of noting the seasonal incidence of some diseases and the epidemic occurrence of others. The number of some diseases are not a little remarkable, but of course hospital statistics in a town like Edinburgh have to be very carefully sifted. In Vienna, for example, nearly all cases go straight to the Allg. Krankenhaus; here a filtration takes place through the dispensaries, and our statistics undoubtedly show an undue proportion of some of the less common diseases. I propose to-day to follow (though I have elsewhere criticised it adversely) the simple alphabetical order, and to refer to those diseases which I consider present points of special interest in the statistics, as well as to those diseases which are at present attracting general interest. In the case of certain of the rarer diseases I am fortunate in being able to show, thanks to Dr Low, a lifelike representation of the condition at the

time of admission, and I take this public opportunity of saying how much the Skin Department and the students of this school owe to his artistic skill.

The first disease to which I think attention falls to be directed is alopecia areata. It is certainly remarkable that out of 2500 cases we should have had no fewer than 100 of that disease. In Croker's statistics, of 10,000 hospital cases 253 were alopecia areata, of 5000 private ones, 291. Private statistics are not so dependable as hospital ones. Of my last one hundred cases, seven were alopecia areata, and in a hundred taken at random there were nine. But though these figures happen to support my view, I think it better to leave them out of consideration. In M'Call Anderson's statistics, published with the report of the Glasgow Skin Hospital just at the time of his death, there were 1860 out of a total of 61,500 [41 years]. If the proportion in London had been as high as with us Croker's figures would have been 424 instead of 253, while the Glasgow figures would have been 2607 instead of 1860. I have not time to-day to go more closely into our statistics, but I may note that the last three months have provided us with no fewer than twenty-nine cases of this disease, almost twice the usual average. Believing as I do in the contagiousness of this disease I am extremely interested in these figures.

I do not think I can do anything more usefully to bring before the Society the scandal of favus in Edinburgh than by marching through the theatre a small detachment of the cases at present under our care. We all knew that favus was a disease which was common in Edinburgh, but I question whether any of you would have believed that we could without difficulty lay hands on nearly one hundred cases of this really terrible disease. In the better classes it is easily cured, but when it gets a hold as it does in the lower classes, it is, under ordinary circumstances, an incurable disease, and the unfortunate sufferers are cut off from connection, and their future is easily prognosticated. Some of these cases have been under our care for three or four years. One of them has had the hair removed no fewer than eight times, and has thus been on the verge of cure over and over again. But we have no control over them, and they disappear when in the opinion of their parents they are nearly well, and only return when the disease has resumed its old grip. I am enough of a Socialist to maintain that it is the duty of the authorities to take these cases in hand and stamp out, as they easily could do, this blot upon Edinburgh, and I hope to

impress this view upon the new Medical Officer of Health for the city.

Herpes Zoster.—Like other dermatologists I have long been struck by the apparent epidemic occurrence of this disease, and I am rather disappointed that my statistics do not demonstrate this fact more clearly. Something of grouping is seen, but not so much as I would have expected. I daresay one reason is that the pain of herpes keeps many people in bed, and it is not so easy to drop into the infirmary for free advice, while another is that many cases go to the medical waiting-room and are admitted to the medical wards. There were altogether twenty-four cases—seventeen in males, seven in females. Four cases occurred on the 2nd and 3rd of February of this year. I would be much obliged if those who have cases in private at this time would inform me of them.

Impetigo Contagiosa.—The figures for this disease pretty clearly demonstrate its epidemic character. The general recent increase is due to the closer supervision now exercised in the Board schools.

I would just like to say that in my experience the main element of speedy success is the thorough application of properly made starch poultices. When a case is not doing well it will generally be found that these are badly made. The figures are as follows :— 1907, January, 7 ; February, 8 ; March, 5 ; April, 10 ; May, 10 ; June, 17 ; July, 12 ; August, 26 ; September, 17 ; October, 16 ; November, 17 ; December, 14. 1908, January, 14 ; February, 24 ; March, 23 ; April, 21.

We have had twenty-three cases of lupus erythematosus. There is nothing special to record of these. As usual they have proved obstinate and capricious in their response to treatment. The most interesting fact I have to record regarding them is in connection with the theory that they are in a sort of indirect way of tuberculous origin. This theory was put forward by the French school just after it had been made sufficiently clear to every one that the disease was essentially a different one from lupus vulgaris. The French theory (held not only in France) is that although the disease is not itself tuberculous, it is nevertheless dependent upon toxins produced by the tubercle bacillus in some hidden focus. Every case of lupus erythematosus occurring in an individual with tuberculous glands, or the evidence of these in the past, was triumphantly held up as a proof of the correctness of this theory. It is not easy to prove a negative. If a patient had lupus erythematosus and any one asserted that this was in some indirect way the result of

tuberculosis, it was not easy to demonstrate that it was not, and one was compelled to wait for the fortunately few cases who died from some other cause while still affected by lupus erythematosus. Some of these have shown tuberculosis on examination, others have not. It has always appeared to me that any case of the disease in which the absence of tubercle could be demonstrated demolished the argument, but this view is not accepted by the supporters of it.

When Calmette's reaction was first made known it did not occur to me to make use of it in this connection and the disastrous results of one of the very few experiments which I made determined me not to make any more. But in Pirquet's reaction we have a test which is, so far as I can see, quite harmless and likely to prove very useful. I show the first case in which I made use of it. The girl had on her hand a condition which I had no hesitation in diagnosing as lupus erythematosus, but on the nose the appearances were such as might be confused with lupus vulgaris. Accordingly, to clear the matter up, she was vaccinated with tuberculin with an entirely negative result.

I have repeated this on one or two other cases. Some have shown reaction, always slight, and others have not. As none of the patients were in the moribund condition in which the reaction is stated not to appear, I am disposed to regard the experiment as on the whole, in favour of those who look on the disease as non-tuberculous, although I know perfectly well that those who hold the other view will direct all their attention to the fact that some of my cases reacted.

I intend to continue the experiment on all available cases, and I shall faithfully record the results of these and of control cases.

It must, however, be kept in mind that a positive reaction does not prove that any particular eruption on the skin is of a tuberculous nature.

I saw not long ago a young lady with an eruption on the face which I had no hesitation in diagnosing as rosacea. She had been under the care of an expert in another city, who, having found Calmette's reaction positive, came to the conclusion that the skin eruption was tuberculous. He then proceeded to treat it with considerable doses of tuberculin with at first distinct improvement. I have little doubt that there was in that case some hidden tuberculosis which was responsible for the reaction while the improvement which followed injections was due to the improvement of the general health or was possibly a mere coincidence.

Lupus Vulgaris.—I do not propose to weary you with details of treatment or discussion of the number of volts, etc., which ought to be used in connection with X-rays. But I think it will be of interest that I should demonstrate some cases which have been long under treatment in the Skin Department. As I have said elsewhere, I think the chief service which Finsen rendered to Medicine was in demonstrating anew the merits of perseverance. These cases, the former extent of which can easily be seen from the scars, demonstrate what can be done by that useful quality. I am not, however, sure whether the oleate of mercury which Jamieson has a large share of the merit of introducing to the profession in connection with this disease is not very nearly as useful as the X-rays, and I am certain that Hutchinson's old method of boring out nodules with a match dipped in the solution of the acid nitrate of mercury will cure a great many cases. It is the combination of two or more methods that usually leads to success, and I am no blind adherent of any one means. In connection with X-ray treatment I, some years ago, drew attention to the greatly increased proportion of cancerous developments in cases which healed under treatment by this method for long periods. Similar observations have been made in other quarters. Sequeira found that nearly 3 per cent. of his male cases of lupus developed epithelioma, and Dacosta had seven cases out of seventy-two treated by X-rays. I have no time at present to discuss the connection between the treatment and the application, important though it is; but I wish, in mentioning it, to point out the importance of being on the constant watch for the early symptoms of malignant disease.

It is, I think, generally taught—I speak liable to correction—that carcinoma in young persons is specially virulent. I am, of course, not in a position to speak of general carcinomas, but I must say that it has not been my experience in lupus cancer. I am able to-night to show one case and a cast of another which demonstrate the curability of some at least of these cases. My first case was that of a young lawyer who has been under my treatment for many years. With the progress of his lupus we are not at present concerned. Some four years ago he developed a warty growth, which, when I saw it, had reached the size of a walnut. It was obviously malignant, and I told him that it must be removed. Mr Wallace kindly took him in charge and excised the growth, which, on examination, was beyond all doubt an epithelioma. The condition of the parts around was such that in spite of all efforts the edges could not be brought

together: the wound granulated from the bottom, and ultimately after several months healed up. Very soon, however, the growth reappeared, and, as these patients will so often do, he kept away from me, hoping against hope that it would disappear. When I next saw him the growth was as large as an orange. A surgical operation would have been a formidable one and the after-treatment very prolonged, so, after laying the facts before him, it was agreed that I should treat the growth by erasion. This I did and came very soon down to a firm layer of fibrous tissue, which I thoroughly scrubbed with caustic potash, as recommended by many of the older surgeons. The growth reappeared before he left the home, and in six weeks was nearly as large as before. I readmitted him, repeated the scraping, and applied arsenious acid. The pain of this application was very great, but he bore it most nobly. He was only a week in the home, and when he left the granulations looked healthy. Next time I saw him there was a small recurrence, which was again scraped under local anæsthesia and treated by chromic acid. I am satisfied that the results are at least as good as could have been achieved by any cutting operation, while it would have taxed even the plastic skill of the late Mr Annandale to have made flaps from the lupus tissue.

The patient, whom I now show along with a very bad photograph taken by myself of the condition before operation, was treated at once by erasion and the application of chromic acid with, I think it will be admitted, complete success.

A most interesting case, of which I can only show a cast, was that of a patient who was treated in a provincial infirmary for lupus by the X-rays. She was a very intelligent girl, and made herself so generally useful that she apparently had the run of the place, and illogically argued that if some X-ray treatment was good for her disease then more would be still better. She accordingly, whenever opportunity occurred, and it occurred daily, used to give herself a dose of the X-rays. The cast shows the condition when I first saw her in August last, and I must explain that unfortunately she came up the day that Dr Low was going on holiday, and the cast was taken by myself and painted by him from notes and from memory. The girl came to me prepared to hear the verdict that her arm must be removed, and it was with considerable misgiving that I explained to her mother the whole reasons of delay, and took upon myself the responsibility of treating it by less radical measures. The edges were scraped and the whole surface treated with arsenious acid

paste. She bore the suffering admirably, and the results have been most satisfactory. I saw her last week, when a small recurrence, the size of a small hazel-nut, was present. This I removed under local anæthesia, and I have little doubt that it will do well. She has a perfectly useful right hand.

The first case in which I used the new tuberculin was one of mixed infection (tubercle and syphilis), and the patient was a mere wreck of humanity. Dr Shennan kindly undertook the estimation of the index, which was then supposed to be essential to the carrying out of the treatment. Her improvement was very marked, and I was greatly encouraged to continue with the treatment. But it was soon evident that if every case was to be controlled by the index, only a very small percentage of my patients could obtain the benefit which was so obvious. Accordingly I joined the ranks of the empirics, and unless there were obvious reasons to the contrary I gave all my severe lupus cases an injection of 1.15000th of a milligram every four weeks. In all of them the result has been satisfactory, the most noticeable feature being the way in which the patches break up, strands of healthy tissue appearing among the areas of the disease.

The patient whom I now present was entirely unable to work owing to the extensive lupus of both his arms. He is now able for, and has been offered, work, but he is so impressed with the improvement that he proposes to continue working at the treatment for a time longer. In his case the systematic inunction of oleate of mercury and exposure to the X-rays have been used in addition.

I have not yet commenced the administration of tuberculin by the mouth, but if it is as satisfactory as is reported by Latham, it would seem that we have still further simplification of treatment at our disposal.

Molluscum Contagiosum.—We have had in sixteen months, out of 2500 cases, eleven cases of this disease. In M'Call Anderson's 61,000 cases only thirty-two occurred. Five of my cases were regular attendants at the public swimming baths. The connection between molluscum contagiosum and public baths is well recognised. In the East (not the East of Scotland) it is known as the itch of the bath. It does not by any means occur always on parts which are covered by the bathing-dress. I believe the best method of treatment is to seize each tumour with a pair of forceps and eviscerate it. Ordinary squeezing is useless, for that merely squeezes out part of the

contents, the pressure must be such as to cause the whole tumour to shell out of the skin.

This year I have had a remarkable series of ringworm of the nails. In my book I refer to this as an astonishingly rare affection, and I am still astonished at the rarity of the disease in children, but I am now wondering whether it is my carelessness or coincidence which has brought together in three or four months no fewer than five cases of ringworm of the nails in adults. They are not new cases, some of them have lasted for as much as nine years, and it is deplorable to think how much disease may have been spread innocently by their owners. One of these cases I am able to demonstrate, others though not here in the body are here in the wax, for I can show casts of them either by Dr Low or by my secretary Miss Rae. The fungus of which I hand round the photograph, taken in the College of Physicians' laboratory, is extraordinarily abundant, and one is really at a loss to understand why these patients did not constantly suffer from outbreaks of the disease on other parts.

Of rosacea we had sixty-two cases. Of these, five are noted as suffering from indigestion, three from constipation, two from headaches—one "used to be troubled with indigestion" in another the appetite is noted as poor, and another is alcoholic. In fifteen it is noted that the patients asserted that there was nothing wrong with their digestion, and as the question was generally asked we may assume that at all events the majority of the remainder did not suffer severely.

I am a very thorough believer in the seborrhœic nature of this disease, and over and over again I have had evidence both in hospital and private work which has strengthened my belief. But I hardly hope I to be able to demonstrate the application of the theory so convincingly as I am able to do to-day. The patient whom I show is the original of the illustration in the last edition of my book. She came to the Infirmary on 12th February.

Her card says she has suffered from the eruption for twelve years. She has no indigestion. She has marked seborrhœa of the scalp. She has since carried out treatment for the seborrhœa and when she came to see me on Monday I did not recognise her. Neither I think will you.

She is further interesting to me in that she has typical psoriasis on her knees, but I do not propose to weary the Society with my own fads.

Vaccine Therapy.—Perhaps general interest in skin depart-

ments is at present most directed in the merits of vaccine therapy. I may say at once that in the case of certain diseases the method recalls the Chinese incident of burning down the house in order to get roast pig. There are such simple, such efficient, such safe means of treating the diseases I refer to, that I have not thought it my duty to experiment on them.

In others I am a thorough convert to the new method. In generalised furunculosis there is no doubt of its efficacy, but it is in sycosis that I have found it of most real help. It is remarkable how opinions differ. When in the early days of enthusiasm for X-ray treatment of this disease I felt compelled to enter a protest against the excessive claims put forward I was referred to as something like a dermatological Mrs Partington. And now I find my friend Dr Whitfield, one of the earliest and most scientific of the opsonists, expressing himself with some doubt as to the merits of the vaccine method in this disease. One can only speak as one finds, and I can simply say that cases that have defied all other treatment have yielded to injections to vaccine prepared from their own staphylococci for me by Professor Ritchie. One of these cases I present. His case was so severe that he was about to be dismissed from his situation, and when I tell you that he was not a salesman in a Princes Street shop but a warder in the Calton Jail, you can understand how bad he was. He has had seventeen injections, and is now practically well.

I have used this treatment in two cases of pemphigus foliaceus. One of these cases will be shown later by Dr Low, and, in the meantime, I prefer to reserve my judgment on its merits. At all events it has done no harm.

I think it will be expected that I should say something of this treatment in acne. Briefly my opinion is that in uncomplicated cases of that disease it is not of much value. There are cases of pustular eruptions on the face which masquerade as acne, but are not that disease ; and believing, as I do, that acne is due to a definite organism, I fail to see why one should expect acne to disappear on the injection of staphylococcal vaccine.

I had in my ward a patient with psoriasis, who at the same time suffered from furunculosis. He was treated with vaccine made from his own cocci, and though his boils improved his psoriasis did not.

Meeting IX.—June 3, 1908

Dr JAMES RITCHIE, *President, in the Chair*

I. Exhibition of Patients

1. *Mr F. M. Caird* exhibited two patients.

(a) A boy, æt. 14, when working in a coal-mine, was thrown from a runaway hutch and sustained a severe bruise of the lumbar region. He suffered intense pain, and on the following morning was lying with his legs drawn up: tenderness in the lumbar region and over the right rectus, a rapidly increasing pulse-rate, and somewhat diminished liver dulness. Median laparotomy revealed extensive subperitoneal hæmorrhage into the ascending and hepatic mesocolon and adjacent parieties, a distended cæcum, and rather collapsed transverse colon. There was no other lesion, but the peritoneal cavity was filled with blood-stained serum. The peritoneum was washed out and Keith's tubes used to drain the pouch of Douglas. In view of a possible paresis of the colon, the appendix was brought out through the abdominal wall and a rubber tube passed through it into the cæcum. On the third day, fæces came from the tube and flatus was passed. The bowels then acted after a dose of calomel, the rubber tube was removed, and satisfactory progress followed.

(b) A young man, æt. 23, who had symptoms of perforated gastric ulcer. A large aperture was found at the pylorus, and its closure so narrowed the gastric outlet that a posterior gastro-jejunostomy was also made. He passed flatus on the third day, and the bowels acted. A few hours later he awoke with severe pain, and became collapsed. Pulse, 110; temperature 96 with cold extremities and pinched, greyish face. Chloroform was administered, and on removing the dressing the abdomen was found to be greatly distended and drum-like. A free mesial incision displayed a huge volvulus of the ileum, black, tense, and as it was manipulated it gave way at a small point. Clamps were applied, the entire gut and stomach were emptied, and the gangrenous ileum removed. End-to-end suture and irrigation of the peritoneum was carried out, the abdomen closed, and the pouch of Douglas drained. In all 10 feet 3 inches of gut were removed. The patient has made a good recovery. At the end of four weeks the diarrhœa, which plagued him at first, has ceased, and he has one motion a day.

2. *Mr C. H. Cathcart* exhibited a case of supposed exostosis of lower jaw.

Annie L., age 25, was admitted to Ward 15, Royal Infirmary,

on February 17, 1908, on account of firm closure of the lower jaw. Her symptoms began in 1901, by pain, stiffness, and a crackling sound in the right temporo-maxillary joint on movement. After some weeks she was admitted to the Edinburgh Royal Infirmary, and an operation was performed on the right condyle of the lower jaw (excision ?). For a year she seemed to be quite well, but stiffness and pain attacked the left temporo-maxillary articulation. In July 1904, she was admitted to Glasgow Royal Infirmary, where an operation was performed upon the left condyle. She was only partially relieved, and the stiffness soon returned and gradually increased.

When she was admitted to Ward 15, her mouth was so firmly closed that she could receive fluid nourishment only, and that through an aperture formed by the extraction of two lower incisor teeth.

Injections of fibro-lysin were tried and were followed by some improvement. They had to be abandoned owing to their setting up irritation at the seat of puncture, and the closure rapidly became as firm as ever.

Electrolysis was next tried, but led to so much irritation that it also was given up.

The only resource then seemed to be a modification of Esmarch's operation, performed at the angle of the jaw on one or possibly both sides. This was explained to the patient, and she was not only willing, but desirous, to have it performed.

Under chloroform, a skin incision was made at the angle of the jaw. As the patient's breathing was then somewhat embarrassed, Mr Cathcart refrained from the operation till the breathing should be more satisfactory. In trying to lift up the base of the tongue through the floor of the mouth he noticed that the lower jaw had become relaxed. A gag was introduced and the jaw was widely opened quite easily, and without the sensation of any adhesions having been broken down. As this showed that the closure had—recently at least—been functional and not organic, the operation was stopped at that point. The skin incision was stitched up, and the patient was allowed to come out of chloroform with a gag holding the jaws wide open. This was to impress upon her mind that full opening was possible. Afterwards everything was done to encourage her in the belief that the jaws would give no further trouble. She left the hospital in a few days with full voluntary power of opening her mouth. She has remained quite free from trouble since going home a fortnight ago.

Although this, in its last phase at least, seems to have been a case of hysterical contraction, the patient was not one in whom hysteria would have been expected. The early symptoms were like those of rheumatism. The third surgeon into whose hands she fell discovered the absence of organic mischief only by accident. Whether or not his predecessors were deceived, as he just escaped being deceived, the case is instructive as showing the amount of mutilation that a hysterical patient was willing to undergo to obtain relief from a fancied inability to open her mouth.

II. Original Communications

1. REMARKS ON CONGENITAL STENOSIS OF THE PYLORUS IN THE ADULT

By William Russell, M.D., F.R.C.P.Ed., Physician to and Lecturer on Clinical Medicine in the Royal Infirmary, Edinburgh; Lecturer on Practice of Medicine; formerly Lecturer on Pathology, School of Medicine, Edinburgh.

Pyloric Stenosis

In approaching the special subject with which this communication deals it is not inappropriate that the generally recognised causes of pyloric stenosis should be enumerated. No doubt the recognised causes are usually the common causes. On this ground the cause of stenosis here specially dealt with might be placed amongst the uncommon causes, for it has been sparingly and hesitatingly recognised even as a possibility—and yet it may soon be seen that it ought to be given a place in the common list. However that may be, at present the commonly recognised causes are, 1. ulcer, 2. cicatrix, and 3. malignant disease.

Pyloric Spasm

That any of these three conditions may cause recurring spasmodic contraction of the pylorus, going on to muscle thickening, will not be questioned by any sound clinical pathologist. That the spasm is due to local irritation will be generally conceded; while the further proposition that the spasm is an important factor in occluding the pyloric outlet will be readily accepted by clinicians who have watched such cases with any reasonable measure of interest.

That pyloric spasm may, in addition to the causes just enumerated, be also determined by the direct stimulation or irritation produced by the chemical constitution or mechanical composition of the gastric contents upon the unbroken pyloric wall will not meet with such general acceptance; while probably only few clinicians have confirmed Lauder Brunton's (1) observation that pyloric spasm may accompany migraine, and be but an additional manifestation of an explosive neurosis.

Effect of Pyloric Stenosis.

Without going into details, which are unnecessary, it may be noted that the main results or manifestations of stenosis are those of stomach dilatation or of stomach ptosis. The size and position of the stomach can, as a rule, be easily determined by physical examination; while the recurring vomiting demonstrates the retention in the stomach of material which, under normal circumstances, is readily passed on into the duodenum.

A further and a very important effect of pyloric stenosis is *visible stomach peristalsis*. This, when present, at once excludes the possibility of the dilatation or ptosis being due to muscular atonicity resulting from catarrh, anæmia, or nervous debility. It is only in pyloric obstruction, associated with the retention of considerable power in the muscular wall of the viscus, that this symptom will be noted; and in my experience such a loss of power only occurs in extreme cases, which are either cases in which there has been a long history of gastric trouble, or are malignant, with a relatively rapidly progressive debility and a correspondingly short history. Where definite pyloric swelling is palpable the merest tyro in clinical medicine has no difficulty in ascribing the gastric symptoms to pyloric obstruction.

Congenital Stenosis an Additional Cause of Pyloric Obstruction in the Adult.

From the preceding summary I hope it may be conceded that I approach the subject of congenital stenosis of the pylorus in the adult with sound views regarding pyloric stenosis in general, and I therefore turn now to the special theme of this communication.

The best way of expounding my subject is to begin from the historical side; and here we need not too seriously concern ourselves with the question as to who recorded the first case. John

Thomson [2] ascribed it to Williamson of Leith in 1841. Osler [3] has put it back to Dr Hezekiah Beardsley in 1788; further research may put it back into the still darker ages of medicine. For our present purpose it is not profitable to go beyond the years 1870 and 1885. In the former year, 1870, Heinrich Landerer [4] published the thesis submitted by him for the doctorate of medicine of the University of Freiburg. It was entitled *Congenital Stenosis of the Pylorus*. After referring to the hitherto known causes of pyloric stenosis and gastric dilatation he proceeded to show that there was an additional condition to those he had enumerated, namely, one in which the stenosis was congenital. He supported this thesis by giving details of ten instances in which he noted pyloric narrowing. The first case observed was a man of 45 years, who had been for years in the hands of doctors on account of stomach disorder. After death it was found that he had an enormously dilated stomach without structural change, no thickening at the pylorus, and yet a pyloric outlet so small as to measure only 2 mm. This led to the careful examination of the condition of the pylorus in other bodies. The result was that he collected other nine instances of narrowing in persons ranging in age from 43 to 63 years. The degree of stenosis varied. Although it was only in the first case that a previous history was available, his clinical acumen led him to realise that this was a condition which must be of importance to the clinician.

In 1885, Professor Rudolf Maier [5] of Freiburg published in Virchow's *Archiv* a contribution to the same subject. He gave a short account of 31 cases in which he had found a pyloric stenosis which he believed to be congenital. The observations were made at *post-mortem* examinations, his attention having been drawn to the matter by a striking example of the condition having occurred in one of his patients. The cases varied in age from 12 to 75 years. The degree of narrowing of course varied considerably, but in none was the narrowing associated with what we would call a coarse structural lesion. As the result of these observations he separated his cases into two groups. First, a simple form; second, a complicated form. The second group contained the cases in which, in addition to stenosis, there was thickening of the pylorus. With regard to the first group, where narrowing was present without other change at the pylorus, he did not think there was reasonable room for doubt that they were congenital; those in which there was pyloric thickening in addition to narrowing he examined more fully and argued more closely in support of their congenital nature.

This argument we need not follow now, for later developments have strengthened his contention that a combined form is met with in the adult. At the same time the subject cannot be left without indicating that the description of the changes he observed will be found well worthy of attention from those specially interested in the exact anatomical appearances presented by them.

Congenital Hypertrophic Stenosis in Infants.

The next important step was the recognition that the condition now known as *hypertrophic pyloric stenosis in infants* was a pathological fact and a clinical entity. Hirschsprung (6) in 1888 seems to have been the first to describe the condition; but many members of the profession probably first had their attention drawn to the subject by John Thomson's (7) paper published in 1897. Since those dates the flood of literature on the subject has attained portentous proportions. It is no part of my present purpose to deal with that literature, or with the differences of opinion which have risen up around the etiology of the condition—that may be left in the hands of the protagonists already in the field. It may, however, he taken as established that there is met with early in infant life a stenosis of the pylorus due to, or accompanied by, marked thickening of the muscle of the pylorus; that the condition is commonly regarded as congenital, and is described as congenital hypertrophic stenosis in infants.

It does not appear, so far as a cursory look through some of the literature has disclosed, that any other form of stenosis has been observed in infants; that is to say, there are no examples of the class Maier called simple stenosis.

The hypertrophic stenosis is much the same as Maier's *Combined Form.* It is so easy of recognition in extreme cases that one wonders now that clinicians had not recognised it long before.

On that lack of observation on the part of clinical pathologists it is not necessary to enlarge. It is, however, necessary to claim that in a condition of this type there must of necessity be degrees of stenosis and degrees of thickening. All infants with hypertrophic stenosis of the pylorus do not die. Only the severe cases are seen by the expert, and it is only for them that surgical aid is required.

That all cases have not the same measure of pyloric narrowing, and therefore do not show equally urgent clinical symptoms, will presumably be granted as a sound proposition. In support of the

proposition there indeed emerges from the literature what to my mind is an illuminating difference of opinion. It is this. Controversy has arisen as to the part that spasm of the pylorus takes in the production of the hypertrophy and of the stenosis. Pfaundler (8) goes far when he holds that the condition is entirely one of local cramp or spasm, and that it can be successfully combated by medicinal and dietetic measures. Pfaundler's contentions are based on clinical observation, and, as such, they belong to the data on which judgment has to be founded. At the same time, if I do not question his observations, neither do I question the observations of those who have had experience of cases unrelieved by dietetic or medicinal measures, and imperatively requiring surgical measures for the relief of a permanent and structural obstruction.

From this it must be inferred that there are degrees of stenosis, and that a pyloric stenosis is not incompatible with infant life; that in spite of its presence life may be continued into childhood and even onwards.

Simple Stenosis in Infants

That, so far as I know, there is no recognition in infancy of what Maier calls simple stenosis is to me inexplicable, and when cases begin to be recorded of simple stenosis of the pylorus being found in children of 6 years when on the operating table, it is at least suggestive that there is a clinical hiatus which ought not to be difficult to fill.

The Present Medical Position.

When we inquire into the position congenital stenosis of the pylorus in the adult occupies in the realm of clinical medicine at the present moment, there are facts to be noted which have considerable significance. The article on dilatation of the stomach (9) in Allbutt's "System of Medicine" is from the pen of the erudite editor himself, but there is no mention of this condition amongst the causes of dilatation.

Sidney Martin (10) and Hemmeter (11) do not mention the condition. Ewald (12), quoting Landerer and Maier, includes congenital stenosis amongst the mechanical constrictions met with at the pylorus. Van Valzah and Nisbet (13) recognise congenital pyloric stenosis in infants, and state that, with the observance of a proper diet, they may live into adult life.

Lambert and Fisher (14), writing in 1907 on benign stenosis of the pylorus, do not mention the congenital form.

Riegel (15), writing this year, only refers to congenital narrowing of the pylorus as due to muscular hypertrophy of the pyloric sphincter, and as leading to early death.

Boas (16), writing in 1907, merely mentions congenital pyloric stenosis in the adult, and passes on to the consideration of other causes of stenosis.

Osler (17) asks if some of the cases of dilated stomach with thickening or hypertrophy of the pylorus may not be congenital.

The Present Surgical Position.

When we turn to the surgical side of the literature dealing with the abodmen, the following may be quoted :—

Cautley and Dent (18) refer to three cases as suggesting a congenital origin, occurring at the ages of 6, 11, and 22.

Mayo Robson and Moynihan (19), after reviewing and discussing the literature on congenital hypertrophic stenosis, conclude as follows : " There can be little hesitation in affirming that a congenital abnormality of the pylorus of some, at present indeterminate, character may after the lapse of few or many years be so altered, or added to, as to cause symptoms of pyloric obstruction. The conditions and the frequency of such cases require further investigation." Mr Mayo Robson, in his large experience, had only operated on one such case at the date of writing, the patient being a young man of 24 years.

Maylard (20), of Glasgow, in 1903 wrote a paper that has not received the attention in this country to which it was entitled, on Congenital Narrowness of the Pyloric Orifice. The paper is based upon observations made when the abdomen had been opened for " chronic gastric derangement." The condition of the pyloric orifice in those cases was not only sufficient to explain the symptoms which existed, but " to suggest that the condition itself was probably of congenital origin." In 1904 he recorded another case, a man aged 31, in whom at the time of operation he found a hypertrophic stenosis of the pylorus, which he believed to be congenital, presenting the " funnel-shape " type described by Maier.

In none of the cases recorded does it appear that any attempt had been made at a differential diagnosis before the patient was submitted to operation.

When Landerer and Maier wrote, the surgery of the abdomen had not entered upon the brilliant operating epoch included in the last twenty years ; and although they both insisted on the great

value of differential diagnosis, the success of surgical operations on the stomach may be said in the interval to have dulled the desire for diagnostic precision. That phase has already nearly passed

The Existence of the Condition

From all that precedes it seems to me that the occurrence in the adult of congenital stenosis must be accepted as an established fact. From the observations of Landerer and Maier and from the experience of Maylard it might even be predicted that as soon as attention is definitely directed to the condition of the pylorus cases of pyloric narrowing will be found to be a fairly common cause of the more intractable gastric symptoms To again quote Maylard "There exists a considerable class of patients in young adult life who owe their chronic gastric trouble to a congenital narrowness of the pyloric orifice." That both the simple and the hypertrophic forms will be found also seems to be probable.

Three Cases.

As a modest contribution to the diagnosis of congenital simple stenosis of the pylorus I submit the following three cases which have fallen into my hands in the last two years.

CASE I.—Thomas C., aged 34, married, was admitted to the Edinburgh Royal Infirmary on August 28th, 1906. He complained of vomiting and pain in the abdomen, and stated that he had been very bad for the preceding six weeks On going into his history it was ascertained that he had been troubled with his stomach for fifteen years, that is, since he was a youth of 19 years The general description of his symptoms was that he had discomfort, fullness, and heaviness in the stomach, coming on usually about an hour after food. The discomfort was sometimes followed by, and relieved by vomiting Sometimes the vomiting was at night The vomit when copious, he thought, consisted of what he had taken for some days The vomit had sometimes been dark in colour and frothy Twelve years ago he was treated in the Infirmary for dilated stomach, and for the same condition two years later Since that time he has been troubled off and on with his stomach During the past year he has had comparatively little trouble until six weeks before admission, when discomfort, pain, and vomiting came on The discomfort and vomiting became very severe, and he was confident worse than ever before experienced He had lost flesh and of course weight Constipation had been extreme the bowels only moving once a week if left to themselves There was no history of malaena, nor of vomiting recently poured out blood The patient

had a fresh complexion, but was very lean. The stomach was greatly dilated; the fundus was at the level of the fifth rib, and the lower border below the level of the umbilicus. The lesser curve was lowered in position, while the pylorus was high up under the liver edge. He on several occasions vomited as much as 3 pints, and as much had been removed by the stomach tube. The total acidity of the gastric contents was high, and there was much free hydrochloric acid. From the symptoms and from the stomach dilatation and ptosis there was no doubt about pyloric obstruction. The chemical examination of the stomach contents excluded malignant disease, and the symptoms were not those of gastric ulcer. From the high position of the pylorus I inferred that it was probably held up by old adhesions, and that the fixity with the addition of ptosis would so kink the outlet as to lead to obstruction. The patient had a gastro-enterostomy performed by Mr Caird. At the operation no adhesions were found, and there was no evidence of recent or of old ulceration. The difficulty at the pylorus was due to an indurated and apparently fibroid condition of the pyloric ring. The patient made a speedy recovery, and was entirely relieved of his symptoms. It never occurred to me that the condition of the pylorus found in this patient might be congenital until Mr Maylard, of Glasgow, wrote to me on the subject, pointing out that the condition corresponded with cases he had described. The suggestion at once appealed to my judgment, and I was surprised it had not instinctively occurred to me before.

Case II. — James M., aged 42, married, was admitted to the Edinburgh Royal Infirmary on July 22nd, 1907. He complained of vomiting, constipation, giddiness, and weakness. He stated that he had lost much flesh during the preceding fortnight, and he was so ill that he had been a week in bed before admission. Careful inquiries into his history elicited the fact that he had suffered more or less from his stomach all his life. Even when he was a herd-boy he used often to vomit. The vomiting attacks came on at intervals of a few weeks, at other times after an interval of a few months. The attacks were preceded by "sickness" (nausea). After vomiting he was relieved, but the vomiting might continue for some days, but after that he would be all right again for a while. When asked about the character of the vomit, he said that he "always minds that it was very, very sour." At that time his food was the same as his brothers and sisters had, and they did not suffer as he did. These attacks of stomach disorder had recurred all through life, and it was only by great care as to his feeding that he was able to avoid their more frequent occurrence. He usually breakfasted at 9 A.M., and could not take another meal until 2, 3, or 4 P.M. His afternoon meal commonly consisted of rice-soup made with mutton, some of the mutton being cut up finely and put into it, or of fish

or tripe with bread. He gave up taking potatoes and all other vegetables years ago, as they "always came up." He drank a good deal of skimmed milk and buttermilk. His symptoms had been severe since the middle of June; they were of the same type as formerly, but were more persistent and he could not depend on any meal staying down. The stomach showed dilatation and ptosis, the greater curve was considerably below the level of the umbilicus, while the lesser curve reached the level of the umbilicus. The pylorus was in the right hypochondrium, not far from the costal margin, there was no thickening. Handling the abdomen set up visible gastric peristalsis. The gastric contents showed abundant free hydrochloric acid. The diagnosis in this case seemed to me to be perfectly clear, and I strongly advised the patient to submit to operation. A gastro-enterostomy was performed by Mr Dowden. There was no evidence of recent or cicatrised ulcer; the pylorus had to my eye much the same appearance as the former one, and Mr Dowden could only put the tip of a finger into, not through, it. This patient also made an excellent recovery.

Case III.—Mrs H., aged 51, was admitted to the Edinburgh Royal Infirmary on October 25th, 1907. She was a small, spare, ill-nourished woman. Her complaints were referred to the stomach. She had suffered all her life from indigestion, but for the past four years her symptoms had been more pronounced. In fact during that period her medical history was a varied one, although gastric symptoms, as pain and vomiting, appear to have predominated. She had never been able to eat vegetables; there was a history of having vomited material like coffee grounds shortly before admission to the infirmary. On examination the stomach showed dilatation and ptosis, both curves being displaced downwards, the greater one reaching to considerably below the level of the umbilicus. There was often visible gastric peristalsis. There was no thickening of the pylorus. The gastric contents contained no free hydrochloric acid. From the dilatation, ptosis, and peristaltic movements there was no doubt as to the existence of pyloric difficulty, but without pyloric thickening. The symptoms were not those of gastric ulcer. The absence of thickening at the pylorus led me to negative malignant disease notwithstanding the absence of free hydrochloric acid. In fact, the symptoms seemed to me to be due to the retention of stomach contents; and in view of the life-long history of indigestion and the need of extreme care with regard to her food and great moderation in the quantity taken I looked upon this case as another example of congenital stenosis. I advised this patient to submit to operation. Mr Hodsdon operated. The case was, however, an unlooked-for disappointment. The condition at the pylorus was as I had judged it to be; but there was in addition a malignant growth high up at the fundus, which could not be removed. A gastro-

enterostomy nevertheless was performed and gave the patient complete relief from her gastric symptoms. She made an excellent recovery, but the operation was followed by that curious feeling of "having no mind" which is one of the occasional and mysterious sequelæ of such operations. There was no constant pain, no pain on the ingestion of food, no difficulty of deglutition, no mass to be felt, to guide us to the suspicion of malignant growth at the fundus. The absence of free hydrochloric acid suggested malignancy, but being satisfied that the pylorus was not malignant, the idea was set aside. I concluded that the absence of free HCl was an individual peculiarity which is not rare.

I heard of this patient in the beginning of June 1908. There had been no return of the gastric symptoms and no further signs of malignancy. The mental condition had not improved.

Diagnosis.

The diagnosis may be briefly summed up under the following heads :—

History.—There is a long history of stomach trouble, or of such digestive difficulties that the patient has learnt to be very careful as to the character and the quantity of food taken, and to allow a sufficient number of hours to separate meals. On the intelligence with which diet has been regulated depends the further history. There may be a history of supposed bilious attacks dating back into childhood. In Case II. the patient as a boy had at varying intervals of time attacks of vomiting, which lasted for a day or two. He had had these when his feeding was his share of the food provided for the family, yet none of his brothers or sisters had similar attacks. As he grew to manhood he had learnt great abstemiousness in eating and drinking, and thus saved himself from a frequent repetition of acute digestive disorder. In Case III. the patient had similarly learnt by experience how to regulate her diet. In Case I., where possibly the stenosis may have been less pronounced, there was a definite history of preceding attacks of severe digestive disorder with stomach dilatation. In all three patients previous attacks had been overcome by ordinary dietetic and medicinal means ; they came to hospital because ordinary means had failed, and ordinary means failed in our hands also.

Symptoms.—The symptoms are the symptoms of stomach dilatation or of gastric dyspepsia, vomiting only occurring in the more severe attacks. The attacks of stomach disorder tend to become intensified as they are repeated ; and, as Mayo Robson has

observed something happens, often not till life is fairly advanced, which brings out the pyloric difficulty and leads to all the symptoms of pronounced pyloric obstruction.

Physical Examination.—At the stage when these cases are likely to come to hospital stomach dilatation is easily made out on physical examination. In 2 of my 3 cases gastric peristalsis could be induced. In Case I. the dilatation and gastric atony were too pronounced to allow of this sign being elicited. When gastric peristalsis can be induced the pyloric end of the stomach also contracts and is easily located. In my 3 cases there was no permanent—that is to say, continuous—pyloric thickening. The cases being examples of the simple form accounts for the absence of this sign, whereas in the combined or hypertrophic form there is permanent thickening, although the degree of thickening will vary from time to time. The absence of continuous pyloric thickening is a most important diagnostic point, but the negation of course assumes that the pylorus can be located through the parietes.

Pyloric Spasm or Cramp.—This as a physical sign requires a little special reference made to it. Kussmaul [21] appears to have been the first to draw attention to it. It is recognised by Ewald, Hemmeter, Van Valzah and Nisbet, and Boas. It is a condition that I knew clinically long before I knew that Kussmaul had noted it. When spasm occurs the pylorus hardens and thickens; when spasm relaxes the pylorus softens. The two phases can usually be easily followed by the hand placed over the pylorus, as the patients are lean with a lax abdominal wall. The spasm can be caused by the irritation of the stomach contents, by structural lesion at or near the pylorus, or even by nervous disorder. Spasm occurs both with and without permanent hypertrophic or hyperplastic thickening of muscular and mucous tissue. Such permanent thickening is also no necessary accompaniment of pyloric narrowing; in fact, in my three cases there was no thickening, and reference has been made to similar observations by others. When spasm is present there is thickening, and in this way spasm often enables us to locate with certainty the position of the pylorus, while the knowledge thus gained of its position may enable us to definitely determine after spasm has relaxed whether structural thickening has remained. When structural thickening remains, the question of its cause remains to be decided.

Chemical Examination of Gastric Contents.—The chemical examination of the stomach contents, especially the determination of the

presence or absence of free hydrochloric acid, should, of course, never be omitted, for the result materially influences the formation of an opinion.

Summary.—The diagnosis in the case of simple stenosis is determined by the existence of pyloric difficulty, the character and duration of the history, the absence of permanent pyloric thickening, the presence of free hydrochloric acid in the stomach contents, the exclusion of ulcer, of malignancy at the pylorus, and of a history that would fit in with cicatrix from previous ulceration. When there is permanent thickening the *benign* causes, when we exclude ulcer, are congenital hypertrophic stenosis, and cicatrix from healed ulcer. The field for differential diagnosis can thus be greatly circumscribed and simplified. In fact, once the possibility of congenital stenosis occurring in the adult enters the clinical field of vision, it will, I think, be found to be quite within the differential diagnostic skill of the physician.

Treatment.

The treatment might be summed up in the words of Lambert and Foster (22) when discussing benign stenosis of the pylorus, namely: 1. Control excessive secretions, 2. reduce pyloric irritability, and 3. increase the muscular activity of the stomach wall. Space does not permit of these principles being elaborated here. When dietetic and medicinal measures are no longer sufficient to enable a patient to nourish himself, the physician can, fortunately with confidence, advise the patient to submit to surgical operation.

The Clinical Pathology.

I have not had the opportunity of examining *post-mortem* the character of the anatomical change at the pylorus in simple stenosis. The changes in both the simple and the hypertrophic form have, however, been dealt with by Maier and Maylard. In the three cases which have been in my hands I have only seen the pylorus when it was exposed at the time of the operation and when the surgeon was satisfied as to the smallness of the opening and, at the same time, of the absence of ulcer, new growth, or cicatrix to account for it. Under these circumstances, I fail to see what view the clinical pathologist can hold but that the narrowing or stenosis is congenital. There is, so far as I know, no pathological process which produces this kind of change, while I am told that a more or less

corresponding congenital condition occurs at the internal os of the uterine cervix, and a congenital tightness of the prepuce is common. That the condition may, in some instances, be the true cause of the attacks of indigestion or dyspepsia seen in young people has to be kept in mind, for until the possibility of occurrence is present to the mind it cannot be diagnosed. In young or later adult life symptoms are more likely to become pronounced, for it is a common experience to find digestive disturbances emerging during this period. It must also be acknowledged that, if congenital narrowing be present, gastric catarrh, hyperchlorhydria or pyloric spasm will all tend to present exaggerated symptoms. When, therefore, any one of these conditions is present, the definiteness of pyloric difficulty may be a surer guide to the existence of the congenital defect than we as yet know. Another factor which Maylard lays stress on, probably rightly, is that any condition which weakens the muscular coat of the stomach, such as anæmia or general debility, will bring out symptoms of digestive trouble, owing to the pyloric narrowing effectively preventing the passage of the gastric contents under the lowered pressure of weak stomach contraction. There are thus several explanations to account for the symptoms of congenital stenosis first appearing prominently in adult life, or at least assuming such prominence as to send the sufferer to seek medical help.

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2. TREATMENT OF ANEURISM BY ELECTROLYSIS

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Many attempts have been made to utilise electrolysis in aneurisms, especially in those thoracic ones that are not amenable to ordinary surgical treatment; it has been hoped that the clotting which occurs around the poles might serve as a nucleus for further coagulation and deposits of fibrin, and that the aneurism cavity might in this way become partially filled up. Such attempts have not met with much success hitherto, and the purpose of this research has been to endeavour to determine by experiments on blood serum outside the body what the actual effect of electrolysis is, so far as regards clotting. Various methods of electrolysing the blood in an aneurism have been used by surgeons. Ciniselli introduced needles connected with both poles and reversed the direction of the current every five minutes; of 38 cases so treated 27 were ameliorated but none were cured. In the unipolar method one pole only was introduced and the other was connected with an indifferent pad placed in the vicinity. The difficult question was which pole to introduce. The positive pole gave the firmest clot, but it was thought that it might be difficult to withdraw and that hæmorrhage or even rupture of the vessel might follow. The negative pole gave a large frothy clot made up of hydrogen bubbles, which was not only of little value in setting up a stable coagulation but was thought to be dangerous from the risk of emboli. Dr John Duncan used the bipolar method and introduced both poles, keeping them both well in the middle of the blood stream, but later on he took a further step and gently cauterised the inner wall of the sac with the positive electrode. He was kind enough to write to me the following letter in regard to the results of his 26 years' experience of electrolysis. "In aneurism I have made a new departure which I think promises well. I had found that while

electrolysis might be used with complete success in small external aneurism and in the secondary sacs of aortic aneurism. I did not obtain with it those occasional brilliant cures which had been observed by others. In taking away the risks of hæmorrhage and inflammation of the sac wall, I had also greatly diminished the curative power of the agent. In short, I came to see that the cure had been in most cases due to the very cauterisation of the sac, which had been thought to be so dangerous. I had no deaths, but none of the old sudden cures; I had trusted for cure to the coagulation, the effect of which is slight and had eliminated cauterisation. I determined, therefore, about three years ago, to gently cauterise with the positive electrode the inner wall of the sac, and I have now had two very satisfactory results by this method." Dr John Duncan was therefore of opinion that the coagulating effect of the electrolysis was slight, and that it was necessary to cauterise (really to prick and slightly injure, for no cauterising effect worth mentioning would be produced under the conditions of his experiment) the wall of the sac as well in order to favour the process of clotting. A more recent method is that known as the Moore-Corrado, and it consists essentially in the introduction into the aneurism of several feet of wire and of the passage of a current through the wire into the blood; the other electrode is placed on an indifferent spot. The wire is always made the positive pole for the two reasons already mentioned. The wire used may be of silver, gold, platinum, or iron. Dr Stewart of Philadelphia (*Philadelphia Medical Journal*, Nov. 12, 1898) writes "Silver, gold, or platinum wire is undoubtedly the preferable material. It should be introduced through a thoroughly insulated needle, and be so drawn as to assume several spiral coils inside the sac." Dr Stevenson ("Electrolysis in Surgery," page 101) writes: "In the electrolysis of aneurysms additional advantage is obtained by the needles being composed of steel, for a chloride of iron is formed at the needle be made positive, and in addition to the coagulating effect of the positive pole we have the coagulating influence of the chloride of iron." Professor S. Leduc ("Les Ions et les Medications ioniques," page 28) however, states that "zinc is the best coagulant and very superior to platinum": he showed the writer an experiment of this nature. This experiment has been repeated by me on an extended scale. A large number of electrodes of different materials were prepared and were used to electrolyse blood serum. These are some of the results in tabular form:

	Anode.	Cathode.
Pt. electrodes.	Bubbles of gas which rise en masse	Much gas
Ag. ,,	,,	,,
Ni. ,,	Fair amount of gaseous bubbles	,,
Fe. ,,	Very few bubbles of gas	,,
Pb. ,,	Precipitate forming skin ; a few bubbles	,,
Cu. ,,	Green precipitate ; a few bubbles	,,
Zn. ,,	Firm glove-like precipitate which holds on to the electrode ; no gas at all	,,

An examination of the table tells us that the negative poles all give off gas freely but cause no precipitation.

The positive poles of platinum, silver, and nickel give off gas but cause no real precipitation.

The positive poles of copper, lead, and zinc, cause precipitation, the firmest precipitate being that due to zinc.

With an anode of iron a few bubbles of gas are given off, but no precipitate is formed.

It is evident then that surgeons by using silver or platinum as the active pole have been unconsciously courting the very danger of gas bubbles which they were endeavouring to avoid. We may draw the conclusion that this danger is nothing but a bogie. There is also no advantage in using iron electrodes, pace Dr Steavenson, for no coagulating effect is produced, probably because the protosalts of iron only are produced. Of the heavy metals, Pb., Cu., Zn., which produce a precipitate, the latter is to be preferred, both because its salts if absorbed are less toxic and also because it forms the largest and firmest precipitate and that without any gas at all being given off. In an experiment using zinc electrodes the current strength was maintained at 300 m.a. for 10 minutes, equivalent to 50 m.a. for 1 hour, which is about the dose usually given by the Moore-Corradi method ; during that time the zinc electrode lost 5 centigrammes in weight and a precipitate weighing 5.35 grammes was formed. This precipitate has been kindly examined for me by Dr Drinkwater and by Miss Isabel Mitchell, and they found that it consists chiefly of a loose compound of albumen and zinc, an albuminate of zinc. By using a coil of zinc wire a firm precipitate binding together the wire spirals is formed. It is difficult to say whether such a precipitate in an aneurism would through the deposition of fibrin upon it lead to the coagulation of the blood and to subsequent organisation of the clot, or whether it would be gradually washed away by

the blood stream, or split up by hydrolysis into bodies of smaller molecular weight. The authorities whom I have consulted think that the precipitate would favour the coagulation of the blood. The clinical experience of surgeons and post-mortem examinations would help to settle this question. I venture in conclusion to suggest that surgeons should introduce zinc wire into an aneurism which they may wish to treat electrolytically rather than the wire recommended by Dr Stewart or other clinical authorities who have not tested the coagulating effects of these metals experimentally in the physical laboratory, for my experiments, which confirm those of Leduc, convince me that the passage of an electric current through an aneurism in the orthodox Stewart-Moore-Corradi method in no way aids in bringing about the coagulation of the blood: whatever coagulation is produced is due only to the introduction of the foreign body.

3. CHRONIC INFLAMMATORY ŒDEMA OF THE SUB-MUCOUS TISSUES OF THE NOSE

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Formation and Functions of the Nose

The nasal septum is seldom or never absolutely straight, in fact a straight septum is an abnormally normal one, the outer wall of the nose is thrown into convolutions by the turbinals so as to expose as much secreting surface as possible in the small space available. The inspired air entering the nasal chambers thus strikes against the septum or turbinals before passing back to the naso-pharynx, and is warmed, moistened, and filtered to some extent from dust particles and bacteria before it reaches the cavities of the pharynx and larynx. The nasal mucous membrane is likely therefore, to be a very active structure, it is well supplied with blood vessels, and in certain parts, notably in the inferior turbinal region these vessels take part in the formation of erectile tissue: the size of the nasal cavities can thus be regulated according to the state of the air entering the nose, the inferior turbinals swell up in cold weather or in dusty atmospheres, excitement also influences the state of engorgement of the turbinals, and the well-known phrase, "open mouthed with astonishment" would be more accurately replaced by "shut nosed with astonishment". The size of the inferior turbinals may also be seen to vary during the short period covered

by an examination of the anterior nares, and we are probably all familiar with the fact that if we lie on one side in bed the nostril on that side tends to become blocked.

Method of carrying out the Present Investigation

The number of specimens examined was 70; of these 22 were inferior turbinals, of which 6 were normal, 6 in a condition of slight œdema, 5 markedly œdematous, and 5 in the condition known as "cauliflower hypertrophy." This state, however, is merely one of very extreme œdematous infiltration. Eighteen specimens of the middle terbinal were examined, in addition to 30 which formed the subject (1) of a previous paper. Of the middle terbinals 3 were normal, 10 showed slight œdema, and the rest marked œdematous hypertrophy, or, in other words, nasal polypus formation. In all but a few cases the tissues were removed during life, washed in water to remove blood and mucus from the surface, and placed at once in 5 per cent. nitric acid in spirit. After a week they were transferred to 10 per cent. nitric acid in spirit and changed three or four times according to the size of the specimen. The time required for decalcification varied in specimens which contained apparently an equal amount of bone. The tissues were carried through in the usual way and embedded in paraffin and cut often in serial sections. It was found by no means easy to get good sections of tissues of such varying degrees of density as the turbinals, containing bone, periosteum, loose connective tissue glands, and delicate mucous membrane. The staining method used was the ordinary one—hæmatoxylin and eosin, but Unna's orcein method was used in addition in almost all cases for elastic fibres.

Sites of Chronic Inflammatory Œdema in the Nose

(A) *The Septum Nasi.*—(1) Anteriorly in the region of the tubercle; (2) posteriorly. Swelling is often seen by posterior rhinoscopy, apparently in the region of the posterior border of the vomer high up towards the roof of the choanæ, giving rise to an appearance that may be compared to the "ace of hearts." This is probably merely an extension backwards of the œdema in the region of the tubercle.

(B) *Middle Turbinal.*—The anterior end, lower border, or outer surface are most frequently affected; these parts are the most dependent.

(c) *Inferior Turbinal*.—The anterior end is affected rather more often than the posterior end, but frequently the anterior end, inner surface, and lower border, as well as the posterior end, are all affected.

Normal Structure of Inferior and Middle Turbinal Bodies

Inferior Turbinal.—(Fig. 1). This consists of a bony framework with an irregular surface; within this we find so-called marrow spaces lined by delicate connective tissue and containing a central thin-walled blood-vessel. The periosteum consists of two or three layers of elongated cells, and just outside this there is a dense layer of elastic tissue from which strands of elastic fibres run out radially in all directions towards the mucous membrane (Fig. 7). Beyond

Fig. 1

the periosteum we have a layer of fairly dense connective tissue containing the larger blood-vessels and some unstriped muscle-fibres, and outside this again the erectile tissue-layer made up of thin-walled blood-vessels of irregular size and shape; the erectile tissue is better marked at both ends and along the inferior border of the turbinal. Just under the superficial epithelium and its basement membrane we have the racemose mucous glands (Fig. 2), the ducts of which open through the basement membrane usually into smaller hollows on the surface. Immediately under the basement membrane there is a thin layer of elastic fibres running parallel to the surface but connected with the fibres that run out radially from the periosteum; the whole turbinal has thus a framework of elastic fibres which causes it to return to its normal size when vascular engorgement is over. The covering layer consists of ciliated columnar epithelium, beneath which are several layers of cubical or irregular cells situated upon the basement membrane.

Blood supply.—The larger vessels run near the periosteum more or less parallel to the long axis of the turbinal; from these smaller vessels run radially outwards to the blood sinuses and to the mucous

FIG. 1.—NORMAL INFERIOR TURBINAL: the body and tip of the turbinal have been removed, and the figure shows the large basal cavity in the cancellous tissue. The blood space has been cut across at many places and most of the connective tissue and glands just under the basement membrane and the superficial epithelium (about ½ inch). Note the loose and collapsed character of the tissue, a result marked in many parts.

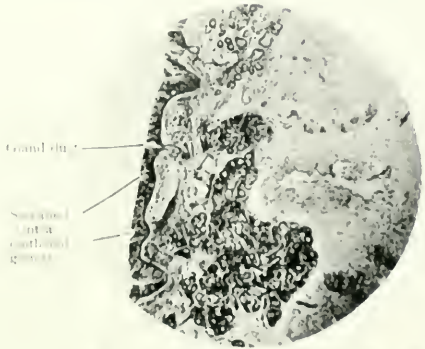


FIG. 2.—NORMAL MUCOUS MEMBRANE of inferior turbinal. Note superficial epithelium containing what appear to be intra-epithelial glands; the appearance may be due to oblique section of a gland duct. Small cell infiltration in and superficial layer between gland and epithelium

FIG. 3.—EARLY INFLAMMATORY ŒDEMA of the inferior turbinal; superficial epithelium is absent; note that the glands are slightly pushed inwards towards the bony core of the turbinal. The œdema of the septal surface of the turbinal is shown when contrasted with outer surface where the tissues are almost normal. × 6 diam.

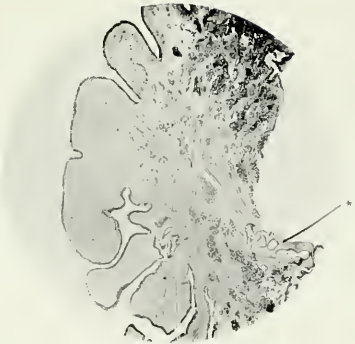


FIG. 4.—Marked chronic inflammatory œdema of the inferior turbinal; "cauliflower hypertrophy"; the glands markedly displaced inwards; dilated ducts are seen opening into the hollows between the papillæ in the lower part of the section; the blood spaces are few and small; the bony core of the turbinal is, however, quite healthy; the walls of the blood-vessels appear to be considerably thickened. × 6 diam.

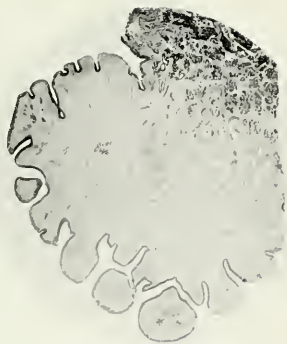


FIG. 5.—Marked chronic inflammatory œdema of posterior end of inferior turbinal. Notice œdema of superficial layers and displacement of glands and blood spaces. × 6 diam.

In all cases the microscopic section has been made in the coronal plane

PLATE VII.—TO ILLUSTRATE DR J. S. FRASER'S PAPER.

Fig. [illegible] Normal middle turbinate. Note the more delicate structure of the middle turbinal the glands are numerous and quite near to the surface, the blood spaces are small and fewer than in the inferior turbinal.

6 diam.

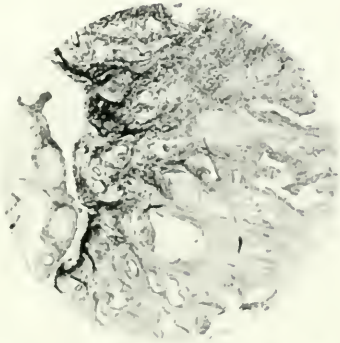


Fig. 7 [illegible] tissue of the middle turbinal. Around the periosteum the fibres form a dense network from which more delicate strands radiate out into the substance of the turbinal surrounding the blood spaces and glands; just under the basement membrane there is a delicate layer connected to the radiating fibres [illegible] 50 diam.

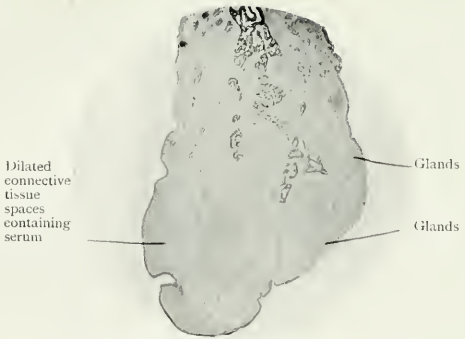


FIG. 8.—Early CHRONIC INFLAMMATORY ŒDEMA of the middle turbinal, or POLYPOID middle turbinal. Note large connective-tissue spaces containing serum along the lower border of the turbinal; the glands in this region are displaced inwards; the bone is healthy. × 6 diam.

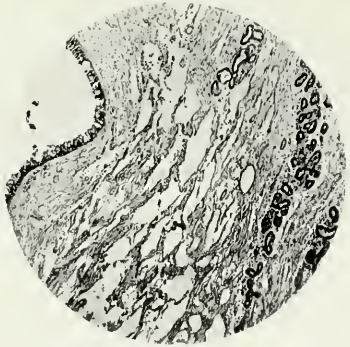


FIG. 9.—Higher power view of same specimen as shown in Fig. 8. Note glands pushed inwards by œdema of superficial layers. × 50 diam.

In all cases the microscopic section has been made in the coronal plane.

PLATE VIII.—TO ILLUSTRATE DR J. S. FRASER'S PAPER

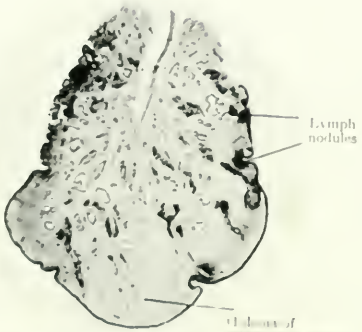


FIG. 10.—Small chronic interstitial abscess of the middle turbinal which has broken through to the nasal cavity. Note the small cell infiltration of connective tissue.

FIG. 11.—Œdema of middle turbinal in case of ETHMOIDAL SUPPURATION. The normal tissues are crowded towards the bone; the thickened lining membrane of the cell in the middle turbinal is well seen, and the small cell infiltration of the mucous membrane.

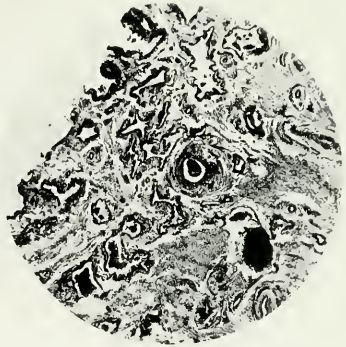


FIG. 12.—This section shows the appearance of the tissues at the base of the polypoid swelling. Note the dilatation of the lymphatic vessels and spaces. In the centre of the field a blood-vessel is seen containing organised tissue—probably a thrombus.

× 50 diam.

FIG. 13.—Section of TYPICAL "NASAL POLYPUS," showing large connective-tissue spaces filled with serum, dilated gland ducts and small blood-vessels the superficial epithelium is almost normal.

× 6 diam.

In all cases the microscopic section has been made in the coronal plane.

PLATE IX.—TO ILLUSTRATE DR J. S. FRASER'S PAPER.

glands, and, finally, very small vessels ramify just under the basement membrane.

Normally in living people there is always a certain amount of small-cell infiltration under the basement membrane and around the glands and superficial blood-vessels; it is only in the unborn that this is not found (Kubo).

Middle Turbinal.—(Fig. 6.) This is much the same as that of the inferior turbinals, but with the following differences: the bone is thinner and altogether more delicate; not infrequently there is a definite cell in the middle turbinal lined with ciliated epithelium, in fact, in these cases the middle turbinal contains one of the ethmoidal cells. The erectile tissue is not so marked in the middle turbinal as in the inferior, and the whole structure is looser and more delicate. From an anatomical point of view we must remember that the middle turbinal is situated higher up in the nose than the inferior.

Microscopical Appearances in Cases of Œdema of the Middle and Inferior Turbinals

(*a*) *The Inferior Turbinal.*—After writing a paper on "The Ætiology of Nasal Polypus," it occurred to me that further light might be thrown on this question by the examination of the inferior turbinal bodies, and I was not surprised to find that the changes leading to so-called hypertrophy of the *inferior* turbinals were exactly similar to those seen in the middle turbinals in cases of nasal polypus formation.

It is necessary in the first place to distinguish between engorgement of the inferior turbinals due to distension of the blood spaces (cavernous tissue) and œdema of the submucous tissue (so-called hypertrophy of the inferior turbinals). This can readily be done by the application of cocaine to the turbinal; vascular engorgement goes down within a few seconds, whilst œdema is little, if at all, affected. In some cases both conditions are present, and, on the application of cocaine, the turbinal shrinks to some extent, but still remains considerably enlarged. It is extremely probable, however, that chronic engorgement is the first stage of inflammation, leading to œdematous infiltration when changes have occurred in the walls of the blood-vessels. In early cases of œdema of the inferior turbinal, the process commences just under the basement membrane, which is frequently thickened (Fig. 3). Kubo considers this thickening to mean inflammation; the leucocyte infiltration is increased,

especially round the vessels and glands, and, in some cases, amounts almost to the formation of lymph nodules. The glands are slightly pushed inwards by the œdema and the ducts dilated; the blood spaces, periosteum, bone, and marrow are not affected.

As the œdema increases (Fig. 4) the glands and blood spaces are pushed further back, and the surface of the turbinal tends to become crenated or scalloped, as seen in sections—papillary, as seen on surface view. This is due to the gland ducts tacking down the mucous membrane at various spots, and can be well seen in the posterior ends of the inferior turbinals by posterior rhinoscopy in cases of so-called "mulberry hypertrophy."

In the œdematous submucous tissue there are a large number of cells showing myxomatous degeneration, and it is due to this fact that nasal polypi were so long regarded as myxomata.

Even in the most marked conditions of œdema of the inferior turbinal—the so-called "cauliflower hypertrophy"—the surface epithelium is but little changed (Fig. 5) and the cilia are well preserved; here and there leucocytes may be seen making their way through the superficial epithelium to the surface; this fact accounts for the muco-purulent discharge present in so many of these cases.

If we examine one of the papillæ on the surface of such an inferior turbinal under a high power, we are at once struck by the close resemblance to a nasal polypus.

The changes in the inferior turbinal are more marked on the inner surface, *i.e.* the surface next the septum over which the air current passes (Fig. 3).

In advanced cases there may be some thickening of the periosteum, but the bone and marrow spaces are always normal so far as my observations go. Finally I have noticed that in these cases the blood spaces are usually small. I have remarked before that they are pressed towards the bony centre of the turbinal by the œdema which occurs between them and the mucous membrane and this fact probably accounts for their diminution in size.

(*b*) *Middle Turbinal.*—The first change noted is œdema of the connective-tissue layer just under the basement membrane (Fig. 8) the connective tissue spaces are enlarged and contain serum, and the glands and blood spaces are gradually displaced inwards towards the bony centre of the turbinal as the œdema increases. The epithelial covering of the turbinal, along with the basement membrane, is of course pushed away from the bony framework.

and later on comes to hang down in the nasal cavity; this is the condition we recognise clinically as "polypoid middle turbinal." The ducts of the glands in many cases become obstructed—on account of the œdematous infiltration—and in this way large cystic spaces are developed.

It is very difficult to speak with certainty as to the condition of the mucous membrane; during the course of hardening, decalcification, embedding, or staining, it often happens that the surface epithelium gets separated and lost; but it is also remarkable that in cases of marked nasal polypus formation the superficial epithelium is apparently quite normal.

Causes of Œdema

Without doubt the two most common pathological conditions affecting respectively the middle and inferior turbinals are nasal polypus and inferior turbinal hypertrophy (so called). Differences in the macro- and microscopic appearances are due to, *firstly*, difference in the structure of the two turbinals, and, *secondly*, difference in anatomical position, or, in other words, the middle turbinal is of looser and more delicate structure and situated further from the floor of the nose.

In both cases the cause of the œdema is to be found in repeated or severe attacks of acute catarrh (coryza): in this condition the turbinals are hyperæmic and slightly œdematous, but as a rule resolution occurs and the parts return to normal: on the other hand in a few cases the inflammation becomes chronic, and the deeper layers of the mucous membrane are involved. Clinically, these chronic cases are characterised by constant nasal discharge (mucous or muco-purulent) and nasal obstruction; the patients complain that they are never free from a "cold" in the head, and they usually state that they have suffered from nasal discharge and obstruction for several years. The tissues of the nose are thus constantly soaking in their own secretions, and a return to normal is prevented. It has been pointed out to me that an analogy may be found in the case of papilloma of the bladder: this condition is so serious because it is impossible to keep the parts dry, whereas warts on the hand tend to shrivel up and disappear when moisture is excluded.

On going over the cases of inferior turbinal enlargement which presented themselves at Dr Logan Turner's clinic during the year

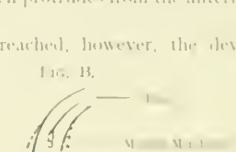
1907, I found that the actual number was 242, but the great majority of these were cases of vascular engorgement. In most of the cases there was only slight enlargement, but it was noted that in 30 there was a co-existing polypoid condition of the middle turbinals, and probably this number would have been greater had cocaine been applied in all instances to the inferior turbinals so as to obtain a better view of the middle turbinal region. Of the 70 cases classified under the head of nasal polypus, there were 20 with co-existing enlargement of the inferior turbinal.

Formation of Polypi

Œdema occurs beneath the mucous membrane along the lower border or outer surface of the turbinal; the mucous membrane thus sags down in the nose, and in advanced cases reaches the floor of the nasal cavities, or even protrudes from the anterior or posterior nares.

Before this stage is reached, however, the developing nasal

Fig. B.



polypus is acted upon by the respiratory air currents, and its base of attachment is specially dragged on in the act of blowing the nose; gravity also tends to increase the formation of the œdematous swelling once it has begun. From what has been said it is not difficult to understand that in some cases the base becomes constricted so that a pear-shaped swelling results, but in my opinion the genuine pear-shaped polypus is much less frequent than is generally believed. I hold that the loop of the snare is frequently responsible for the pear shape, and that in most cases the polypus is broad based along the lower border or outer surface of the turbinal. If we think of the frequency of œdema in inflammation of such loose structures as the prepuce, scrotum, and lower eyelid, we need not wonder that it occurs in the chronic inflammation of the middle turbinal region of the nose. In the inferior turbinal the case is somewhat different; here the tissue is denser, and the lower border

of the turbinal is already almost on the floor of the nasal chamber; consequently we do not meet with such marked œdematous swelling. At the *posterior* end of the inferior turbinal we may, however, in advanced cases, see a large papillary mass of œdematous tissue hanging down in the posterior surface of the soft palate.

Inflammation in any situation is always associated with exudation, and the looser the tissue the greater the amount of exudation. I believe that in early cases of rhinitis we have an excessive amount of vascular turgescence associated with a slight degree of œdematous infiltration; this goes down under cocaine application. If the inflammation becomes chronic the œdema increases most probably on account of changes in the walls of the blood-vessels and blood spaces. Lymphatic and venous obstruction no doubt play a part, and I have observed swelling of the endothelical cells lining the lymphatic vessels in one or two of my specimens. Watson Williams (3) recently showed a specimen of nasal polypus in which a lymphatic vessel appeared to be blocked by inflammatory products. Hopman (4) thinks that the cause of the œdema is some impairment of the circulation in the efferent venous vessels, and the engorged state of the small veins in many of the specimens examined by me supports this theory (Fig. 12). Grünwald (5) explains the œdema by changes in the vessels of the nature of peri- and endo-arteritis, but one is bound to note that even in apparently normal turbinals the vessel walls seem to be abnormally thick. Goodale (6) says, " It is possible that the inflammatory infiltration which penetrates the substance of the turbinals may lead in some places to a stasis from compression of the veins "; the same writer entirely disagrees with the bone disease theory of nasal polypus formation.

In advanced cases the elastic fibres are hard to find—at least in the œdematous parts; near the periosteum they are present and normal in appearance. The striking similarity of the changes in the middle and inferior turbinals will be apparent from the illustrations, and nobody has yet said that inferior turbinal œdema or hypertrophy is due to bone disease; we may well ask why œdema of the middle turbinal should be due to this cause. It is true that the bone is often affected in advanced cases of nasal polypus formation, associated with ethmoidal suppuration, but in this case the bone disease is *the effect*, not the cause (Fig. 11). Yonge's (7) theory a to the causation of nasal polypi was not well received at the last meeting of the British Medical Association at Exeter, and as I have dealt with it in a previous paper I need not do so again.

In regard to Grünwald's theory that nasal polypi are associated with sinus suppuration, I can only state that this appears to be true in only 50 per cent. of cases. I think we are not far wrong if we regard the character of the discharge present as diagnostic—that is to say, if nasal polypi are found associated with a clear watery discharge no sinus suppuration is present; if the discharge is muco-purulent, sinus suppuration may or may not be present; if the discharge is distinctly purulent then sinus suppuration is present.

Morell Mackenzie used to urge against the inflammatory origin of nasal polypi that while nasal catarrh is very frequent in children, polypi are extremely rare. There are two good answers to this objection: (1) the middle turbinal region is not easy to examine in children, and minor degrees of œdema may easily pass unnoticed; and (2) it is extremely probable that the vascular changes leading up to marked polypus formation take years to occur—in other words, children have ceased to be children when polypi are developed.

In conclusion, I wish to express my thanks to Dr Logan Turner, who has kindly allowed me to make use of his clinical material, to the Royal College of Physicians (Edinburgh) for permission to work in their Laboratory, and to Dr James Ritchie for much advice and assistance in carrying out the work.

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(2) *Archiv. f. Laryngologie*, Bd. XIX. S. 202.
(3) *Proceedings of the Royal Society of Medicine*, 1 1908.
(4) *Wiener med. Presse*, 1883.
(5) "Nasal Suppuration," 1906.
(6) "Diseases of Eye, Ear, Nose and Throat," by Posey and Wright, p. 706.
(7) "Polypus of the Nose." 1906.

Meeting X.—July 1, 1908

Dr JAMES RITCHIE, *President, in the Chair*

1. EXHIBITION OF PATIENTS

Dr Byrom Bramwell exhibited a case of OSTEITIS DEFORMANS (Paget's disease). The disease is very rare in Scotland. Osler states (in his last edition, published in 1905) that only about sixty-seven typical cases have been recorded. The notes are as follows:—

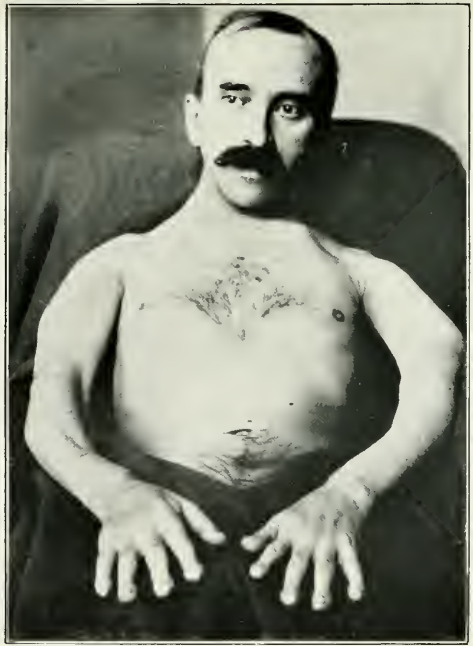

FIG. 1.—Front view of the case of osteitis deformans described in the text.

The bowing of the arms and the enlargement of the ends of the fingers, due to increase of the soft parts, are well seen.

FIG. 2.—The case of osteitis deformans described in the text, seen from the right side.

The marked projection of the left clavicle (which has not been fractured) is well seen.

PLATE X.—TO ILLUSTRATE DR BYROM BRAMWELL'S ARTICLE.

Fig. 3.—Right hand of the case of osteitis deformans described in the text.

The bones of the carpus are almost natural, showing little or no transparency. As one proceeds from the carpus to the metacarpus and phalanges, the bones become more and more transparent, but show a faint bony structure except in the terminal phalanges. In some of the metacarpal bones a distinct medulla can be made out, but in the others a somewhat regular striation of slightly denser bony substance has, in parts, taken its place.

The terminal phalanges, except at the articular ends, have almost completely disappeared.

The head of the metacarpal bone of the index finger of the left hand has been tilted round, and the appearance is suggestive of fracture.

The sesamoid bones in the flexors of the thumb are still present.

Plate XI.—To Illustrate Dr Byrom Bramwell's Article.

L. G. B., widower, aged 43, a forester by occupation, was admitted to the Edinburgh Royal Infirmary on 31st March 1908.

History.—Until the present illness commenced the patient was a well-set-up, robust, healthy man; he had never had syphilis or gonorrhœa.

On 28th October 1903 he tripped when getting into a boat and fractured his left thigh and left humerus. He stated that he was perfectly well up to the time of this accident. *Two months later,* while going about with sticks, he fell and again fractured his left humerus, also his right clavicle and right scapula; in this fall he also strained and hurt his left foot. *Five weeks after the second accident,* the left humerus was again fractured while the doctor was exercising it. *In December* 1905 he slipped while getting into bed and fractured his left forearm. *In September* 1907 he fell and fractured his right forearm and strained his back badly.

Since the present illness commenced (October 1903) he has become dwarfed and stunted. In health, his height was 5 ft. $0\frac{1}{2}$ inches; on admission to the Infirmary, it was 4 ft. 11 inches. His head has become more rounded and larger; the upper part of his face also, it is said, has become wider and broader.

Condition on Admission to the Infirmary.—The patient was unable to stand. There was little or no curvature of the spine and no forward drooping of the head. The bones of both upper extremities were markedly curved like a bow, the elbow-joint being the centre of the bow (see Figs. 1 and 2). The thigh bones, too, were curved forwards and outwards. The hands showed a curious thickening of the ends of the fingers due to increase of the soft parts. The skin of the palms of the hands was remarkably fine, soft and silky to the touch. The clavicles projected forward, especially the left clavicle, which had not been broken. The base of the thorax was broadened out. The abdomen was diamond-shaped with a deep transverse sulcus at the umbilicus. The hair of the body, especially on the legs, had, the patient stated, increased in amount and thickness since the disease commenced. The surface of the affected bones was felt to be irregular and in places nodulated. There was no pain and no tenderness over the bones.

X-ray examination showed marked changes in the bony structure. Dr Hope Fowler kindly made a series of photographs, some of which are reproduced in Figs. 3, 4 and 5.

On admission to hospital the blood count showed (notwithstanding the pallor of the patient's face) an excess of red blood corpuscles

and of haemoglobin in red corpuscles 0,800,000 per cubic millimetre haemoglobin 115 per cent.). The size and shape of the red corpuscles was normal; no nucleated red corpuscles were seen. The white corpuscles were normal in number (7,500 per c.mm.), and the differential count was normal (polymorphs 68 per cent., small lymphocytes 22 per cent., large lymphocytes 6 per cent., eosinophiles 3 per cent., mast cells 1 per cent.).

The urine contained phosphates, a small quantity of pus, and a small quantity of albumen.

Treatment and Results.—For the first five weeks after the patient's admission to hospital he suffered from recurring attacks of fever, perhaps due to the condition of the bladder and urine.

On May 7th suprarenal extract was prescribed, and has been given since. 1 c.c. of 1 in 1,000 solution of adrenalin chloride given hypodermically once a week. Since this treatment was commenced, and presumably as the result of it, steady improvement has taken place. The patient looks and feels very much better; there has been no recurrence of the fever; the urine still contains phosphates, a small quantity of pus, and a small quantity of albumen; and the red blood corpuscles and haemoglobin have fallen to the normal amount as shown by the following table:—

Date.	Red Corpuscles.	Haemoglobin.	White Corpuscles.
2nd April	6,800,000	115 per cent.	6,500
4th "	6,000,000	115 "	6,400
6th "	6,700,000	110 "	6,600
8th May	4,800,000	85 "	5,600
17th "	4,700,000	80 "	—
6th June	4,500,000	80 "	6,800
20th "	4,700,000	80 "	7,400

II. Original Communications.

1. NOTE ON THE TREATMENT OF A TYPE OF SO-CALLED "FAINTING ATTACK."

By HAMILTON GRAHAM LANGHELL, M.D., F.R.C.P.,
Physician to Leeds Hospital.

I have not found it by any means easy to get a title which will describe accurately and yet briefly the subject of the following remarks. I must, however, first intimate that before I have finished I shall have made clear the salient features of the attacks to which I wish

FIG. 4.—Left arm of the case of osteitis deformans described in the text.

On account of the fact that no lens intervenes between negative and area to be radiographed, the resultant prints are always reversed.] Here the bones—humerus, radius, and ulna—have almost entirely lost their normal outline, and have little or no appearance of normal structure. There is only the faintest trace of an elbow-joint. The humerus is altered in shape, outline, and structure; along the outer edges of the humerus there can be seen traces of periosteum and bony tissue, but only in parts. This bone shows evidence of having been broken repeatedly; this is made out where the bending or angling has occurred, and also by the curiously condensed lines running irregularly through the bone. A medullary outline has completely disappeared, its place being taken by irregular patches of tissue, which, I am of opinion, alone indicate the remains of a medulla. The bony substance has been almost completely absorbed.

The radius and ulna are in similar condition, but not quite so advanced. The radius is very nodular; these nodes are made up of soft fibrous tissue from which all the lime salts, etc., have disappeared.

PLATE XII.—TO ILLUSTRATE DR BYROM BRAMWELL'S ARTICLE.

FIG. 5.—Right arm of the case of osteitis deformans described in the text

As contrasted with the other arm, this humerus shows beautifully and at the same time regularly the almost complete absence of bony salts, beginning in the shaft and spreading gradually to the articular ends of the bone.

The ulna shows signs of a gradual bending perhaps synchronous with the evident old fracture of the radius at the junction of its upper and middle third. Both radius and ulna exhibit similar changes in bony structure; where the fracture has occurred there is a little condensation of bony tissue still showing.

PLATE XIII.—TO ILLUSTRATE DR BYROM BRAMWELL'S ARTICLE.

to direct attention, as also the treatment adopted. I shall begin by giving the notes of two cases observed within the last nine months.

Case I.—J. H., æt. 48, has been under my observation more or less constantly for the last twelve years, during which time she has been seen by several consulting physicians. Her symptoms were usually ascribed to her heart, but neither dyspnœa nor palpitation were ever present, nor were any cardiac murmurs or irregularity of the pulse ever detectable. She has frequently alluded to a type of attack which she often took of a somewhat vague character, and until last autumn I failed to grasp the real significance of many of the symptoms of which she complained. Notable amongst these was a marked tendency to a " sinking feeling " constantly following upon evacuation of the bowels. So marked was this that she regularly had to lie down on bed (or a sofa) immediately after stool, and so unpleasant (and almost over-powering) was this sensation that she dreaded (and often postponed) the evacuation, particularly if it was more than usually vigorous, *e.g.* as the result of a purgative. On 12th July 1907 she had been more than usually distressed with what she called " queer feelings " and " fainting attacks," and her pulse had not improved as on former occasions under the use of strychine and strophanthus. Late at night I was hurriedly called, as she had taken a bad " fainting attack " and was thought to be dying. I found her extremely pale, with a rapid pulse of markedly small volume, unable to speak, but acutely conscious of her surroundings. A hypodermic of adrenalin very rapidly improved her pulse, and in a short time she said, " You never gave me anything before, doctor, that acted like that." Having now seen her in an attack I was better able to gather particulars of them. They begin, she says, with a sudden feeling coming over her " as if something dreadful was going to happen," then follows a strange feeling of inward shaking and " sinking " in the umbilical region, unaccompanied by any pain or dyspnœa but with an overpowering sensation as if she was dying, consciousness, however, being always intensely alert, though the power of speaking or even moving is lost. She would fain speak to break, as it were, the spell that she feels overpowering her, but while thought is intensely active the power of utterance is lost. These attacks come on chiefly at night after getting into bed, and only rarely does she have them after getting to sleep. It is interesting to note that she has by experience found that she often goes to sleep most easily in the *prone* position, a feature in her case the

significance of which will appear later. Another interesting condition, she states, occurs frequently if she is awakened suddenly, viz. a feeling as if she "woke up in detachments."—her consciousness wakes quite brisk and vivid, but the power of speaking and moving distinctly lags behind for an appreciable period.

After I had treated her with adrenalin internally for over a month I asked my colleague, Dr Edwin Matthew, to examine her blood-pressure, which he kindly did on two occasions. He reported her *systolic* pressure to be 110 and 120 on these occasions, and this notwithstanding adrenalin had been regularly administered for 5 weeks. On adopting the treatment to be afterwards mentioned her (systolic) pressure was immediately raised thereby 20-30 mm. On discussing her whole symptoms Dr Matthew agreed that it was mainly a want of vasomotor tone which was present in her case, nothing abnormal being detectable in her heart.

Case II.—I was hurriedly called about 11 P.M. on 9th October 1907, to see Mrs B., æt. 38, by her husband, who said his wife had one of her "attacks" and seemed to be dying. The house was fully half a mile off, and when I arrived I found her recovering gradually and able to give me an account of the attack which, with fuller details afterwards obtained, is briefly as follows.—The attacks always come on at bed-time, being most apt to occur after a day's housework involving any special exertion. She has been subject to them since she was about 18, but they have been much more frequent (as also more severe) since she was married 18 years ago). Occasionally the attack would come on while undressing, but usually it was after she was in bed and sometimes just when dropping off to sleep, when an intense feeling of "sinking" would come over her, with a sensation of utter loss of all power and a feeling as if she were dying. Consciousness is always perfectly retained, though, she is quite unable to give expression to her thoughts, which are perfectly vivid. Thus in this last seizure her husband, noticing her pallor, had recognised that one of her "attacks" was impending and made a hurried search for some stimulant to give her, but though the patient realised perfectly what he was looking for (and, indeed, heard him asking her where it was) she was absolutely unable either to tell him or to indicate even that he was searching in the wrong places. He ran off to a neighbour's house for some whisky, and though quite conscious of his errand she wished he had not gone, as she felt so certain she would not live till he returned. Her husband tells me she shows no sign of agitation

in these seizures beyond tears filling her eyes as she (speechlessly) watches his anxiety, but there is no sobbing or even attempt at crying. There have never been any motor phenomena noticed during any of the attacks. The patient is not aware of ever having had an ordinary attack of faint or syncope, and in the seizures she does have there is no sensation of swooning whatever; on the contrary her consciousness is always painfully alert, making their presence all the more alarming to herself. The following day she was in her usual health, and I then learned of a condition of some interest, viz. that for years she had suffered greatly on getting into bed at nights from prinkling and numbness in her hands and feet. She has therefore made a practice for a long time, in order to get to sleep, of doubling herself firmly into the "knee elbow" attitude, so as to benefit by the flushing of the blood-vessels in the extremities which results on relaxing the "tourniquet" action of this "forced flexion." It is of interest to note that she alluded to this habit of hers when I attended her last confinement in December 1906, though I did not recognise the significance at the time. When I was about to leave the house after the confinement was over she asked if she might get on her hands and knees as she could not get to sleep without doing so. Regarding it merely as a foolish notion I forbade her to do so for that night at least, and as she got to sleep all right—much to her own surprise—I thought no more of the matter.

On careful examination nothing whatever in the nature of a cardiac lesion could be detected, nor were there any signs of anæmia; no dyspnœa or palpitation on exertion was present. I was struck, however, with the apparently very low tension of her pulse, and from the various symptoms she described, and my previous experience with Case I., I formed the opinion that the seizures were really of vasomotor origin. My colleague, Dr Matthew, again very kindly took her blood-pressure and reported it to be one of the lowest he had seen, the diastolic being relatively very low, the figures being $\frac{S\ 90}{D\ 65}$

Such is the somewhat imperfect record of the two cases. What can be said as to the nature of the seizures? The symptoms in the two patients are very similar, but that the attack in each was not an ordinary "faint" is evident, for consciousness was painfully acute throughout. The true explanation of these so-called "faints" I would suggest to be this, viz. a rapid withdrawal into the splanchnic "venous pool" of a large amount of blood owing to the dilatation

of the arterioles in this area. In both patients there was distinct (though varied) evidence of defective general vasomotor tone (Mrs B.'s blood-pressure being especially low); but the really important point would seem to be not so much the *actual* blood-pressure of the individual as the liability of those patients (whatever their *normal* blood-pressure may be) to a sudden dilatation of the splanchnic arterioles, with a consequent filling of the large abdominal veins. Founding upon this theory, and acting on a suggestion of Dr Matthew, Case No. 1 (Mrs H.) was ordered to wear a firm abdominal binder with a large pad to keep up pressure over the spanchnic area, and to discontinue the adrenalin which she had been taking with distinct benefit. Since she adopted this measure the patient, though far from cured of many of the symptoms arising from faulty vasomotor tone (e.g. flushings, sweatings, etc.), has never again had any return of these "sinking" attacks. On rare occasions, if she fears an attack may occur, she increases the abdominal pressure by inserting the hand between the binder and the pad, and, lying prone in this position, falls asleep. There can, I think, be little doubt that the "sinking" sensations described above in Mrs H.'s case as immediately following action of the bowels were really minor attacks of similar vasomotor origin, arising from the disturbance of the abdominal pressure. These minor attacks she has also got entirely rid of since she adopted an abdominal binder.

As regards Case II., the practical application of the theory was equally successful. Her attacks seemed to be distinctly produced by the filling of the splanchnic "venous pool" on lying down in bed, consequent upon the removal of the external abdominal pressure kept up during the day by her clothing (especially her corsets). In her case the change produced, as she lay in bed in her ordinary condition, on the volume of her pulse when pressure over the splanchnic area was applied by the hand was most marked. I therefore ordered her to apply a firm binder with pad to prevent the seizures, explaining to her how I expected it to act, viz. as a substitute for her daytime garments. Her remark was most instructive: "Well, doctor, I believe you're right: that must be why I never had any attacks after my confinement so long as I wore my binder. I never required to go on my hands and knees to get to sleep till I left off wearing it." Evidently the pinkling, &c., of her limbs was the result of too low a general blood-pressure when the constriction of her daytime clothes was relaxed.

Dr Matthew agreed that, seeing the blood-pressure in Mrs B.'s

case was so low, it might be well to give a little adrenalin, and five minims t.i.d. were ordered, the abdominal binder being regularly worn at night. After a very few doses, however, the adrenalin had to be stopped owing to the throbbing which resulted. Since then she has had no treatment whatever other than the wearing of the firm binder at night, and not only has she never had another attack, but, as she expressed it, she "feels a different woman," having got rid of the *dread* of the attacks which used to make bedtime a source of terror to her.

General Remarks.

I have been unable to find any clear description of this type of seizure, but Sir Wm. Gowers, in his recent volume *The Borderland of Epilepsy*,[1] describes what he calls "vagal and vaso-vagal attacks," which in several respects resemble what I have described above. Thus he speaks of there being "no impairment of consciousness" in these attacks, and he describes vividly the sense of impending death present in these cases "so intense that no recollection of its falsity in preceding attacks prevents the conviction of its present reality." But he speaks of the patients showing "respiratory distress sometimes so intense as to amount to orthopnœa," as also "cardiac discomfort and acute pain in some cases" . . . none of which symptoms were present in my patients at all. Some of the details of his cases of "vaso-vagal" attack certainly present a distinct resemblance to the two cases detailed above; but, while he does not mention the condition of blood-pressure present in his cases, he evidently regards the condition as one of vasomotor *spasm*, for the remedies he advises strongly are *vaso dilators*. Now the hæmomanometric readings recorded above, it seems to me, are entirely against the view of *this* being the underlying condition in my cases, so that I cannot conclude that the seizures in my patients were exactly of his "vaso-vagal" type. The explanation of them which has been suggested above may be erroneous, but the results of treatment, at any rate, based upon this view appeared to me to be of sufficient practical value to be worthy of record. In conclusion, I desire to acknowledge the kind assistance in the investigation of the cases rendered by Dr Matthew, as also his helpful suggestion as regards treatment.

[1] Gowers, *Borderland of Epilepsy*, p. 18 *sqq*. Churchill, 1907.

2. INTERMITTENT CLAUDICATION OR INTERMITTENT LIMPING AND OBLITERATIVE ARTERITIS WITH ILLUSTRATIVE CASES.

By Byrom Bramwell, M.D., F.R.C.P. Ed., Physician to the Edinburgh Royal Infirmary, etc., etc.

Intermittent claudication is a rare condition, which was first described in the year 1858 by the late Professor Charcot [1] and has attracted a good deal of attention, more especially abroad—in France, Germany and America. In 1898, Professor Erb [2] wrote an exhaustive monograph on the subject. In 1905 Dr J. Ramsay Hunt [3] of New York published an important paper on the subject, in which he detailed the notes of four cases which had come under his own observation, and gave a valuable epitome of the observations of other physicians. Dr F. Parkes Weber [4] has recently published in the *Lancet* a typical case and has referred to the literature of the subject. The obliterative arteritis with which intermittent claudication is usually associated has been exhaustively considered by Mr A. Pearce Gould [5] in his Lettsomian Lectures.

Intermittent claudication or intermittent limping is the term given to a group of symptoms which are due to temporary ischæmia of the affected part. The essential clinical characteristics of the condition are:—

(1) Absence of symptoms while the affected parts are at rest; (2) the development of symptoms (painful limping, etc.) after exertion; (3) the disappearance of the symptoms after rest; and, in most cases (4) absence of pulsation in the pedal arteries. I will presently describe a case of typical intermittent claudication in which there is no arterial obstruction, and no defect in the circulation of the legs or feet.

The Symptoms which characterise an Attack of Intermittent Claudication.—In typical cases, the patient feels quite well until he puts the muscles of the affected part into prolonged action. In some cases, however, more especially in the advanced stages of the disease, weakness, and it may be even muscular atrophy, paræsthesiæ and vasomotor alterations are persistently present (*i.e.*, are

[1] *Compt. rend. Mém. de la Soc. de Biol.*, 1858, Tome v. p. 225.
[2] *Deut. Zeit. Nervenheilk.*, Bd. 13, p. 1, 1898.
[3] *Medical Record*, May 27, 1905.
[4] *Lancet*, Jan. 18, 1908, p. 152.
[5] *Lancet*, March 15, 1902, p. 718.

present independently of exertion. In the advanced stage of the disease, dry gangrene may be developed and severe continuous pain in the affected part may be present.

After walking a longer or a shorter distance, which varies in different cases, motor and sensory symptoms and vasomotor alterations, due it is believed to insufficient arterial blood-supply to the affected part, are developed. The more important of these symptoms are :—weakness, stiffness, heaviness, coldness, numbness, a prickling feeling, pain, cramps, and rigidity in the affected limb or limbs. In consequence of the weakness, stiffness, pain and cramp, the patient limps.

The affected limb usually presents marked evidence of derangement of the circulation. The foot may become red and swollen, or the toes may become white and dead. In the present case, the whole limb, but especially the foot, becomes darker in colour. During the paroxysm the affected limb may be colder to the touch than its fellow on the opposite side.

If the patient rests, the symptoms gradually pass off, and the limb after a time becomes normal.

The condition is said to be the same as Stringhalt in horses.

The development of the symptoms is, as I have already said, believed to be due to defective blood-supply to the affected parts.

In most cases of intermittent claudication, no pulsation can be felt in the pedal arteries. This is an important diagnostic point ; in all cases, in which a patient complains of intermittent limping (weakness, pain, stiffness, cramp, numbness, etc., in the leg) after exertion, the condition of the arterial circulation should be carefully examined.

The absence of pulsation in the pedal arteries may be due to anything which obstructs the vascular channel. In most cases, the obstruction is due to obliterative arteritis or angiosclerosis, which usually affects the small vessels and is, then, often symmetrical ; in some cases, the larger vessels are affected. In some cases, the nerves are involved from affection of the vasa nervorum ; the clinical picture in cases of this kind may be that of peripheral neuritis.

In most cases of intermittent claudication three factors seem to take part in the production of the ischemia which is the fundamental cause of the condition, viz., (1) vascular obstruction, (2) vasomotor spasm, and (3) the increased demand upon the part which muscular

activity entails. But in some cases, the first factor (arterial obstruction) is, as I have said, wanting.

Arrested arterial circulation through the main arteries of a limb does not necessarily produce intermittent claudication. I will afterwards relate two cases which prove this. Whether intermittent claudication occurs under such circumstances or not probably depends upon the condition (freeness) of the muscular circulation and the presence or absence of vasomotor spasm.

I repeat that the symptoms characteristic of a paroxysm in intermittent claudication are due to defective arterial blood-supply (ischemia). So long as the patient is at rest there are no symptoms, at all events in the earlier stages of the case. During rest, the feeble muscular arterial blood-supply is sufficient for the tissue requirements; but during active exertion, the increased demand for arterial blood, which the muscular contractions entail, cannot be met, and symptoms (pain, spasm, muscular weakness, etc.) are consequently developed.

In addition, in some cases, a condition of vasomotor spasm is supposed by Erb and some other authorities to determine the development of symptoms—this brings the case into relationship with Raynaud's disease.

Intermittent claudication and the obliterative arteritis with which it is associated are more common in the male than in the female, and seems to affect the Jewish much more than other races of mankind; one of the cases which I will presently describe (Case II.) occurred in a Jew. The condition may go on for many years. The ultimate effect of the arterial obstruction which is the pathological basis of the condition is the production of dry gangrene.

Anything (such as syphilitic endarteritis, atheroma and non-syphilitic obliterative endarteritis) which causes narrowing and obliteration of the arteries may be the cause, or, in part at all events, the cause, of the condition. In some cases, as I have already said, the large arteries are the seat of the obstruction. In Charcot's first case there was an aneurism of the iliac artery.

Syphilis, alcohol, excessive smoking, exposure to cold, and many other conditions, such as flat foot, the wearing (pressure) of a truss, have been supposed to be etiological factors in the production of the condition. Erb lays special stress upon excessive tobacco-smoking and exposure to cold. In most of the cases of intermittent claudication and obliterative arteritis which have been recorded there was

no history of syphilis, and the arterial disease does not seem to have been syphilitic.

Syphilitic endarteritis obliterans is much more apt to affect the smaller and medium sized than the larger arteries, though the larger arteries, such as the carotid, are occasionally affected. I will afterwards describe a case in point.

The aorta is of course frequently affected by syphilitic disease, and its nutrient vessels are then frequently the seat of endarteritis obliterans. As I suggested many years ago, this obliterative affection of the nutrient arteries of the aortic wall is probably an important factor in the production of aneurism, and explains the weakening of the aortic coats and the production of aortic aneurism as a result of syphilis.

In syphilitic endarteritis, the lesion (thickening) of the inner coat and the narrowing of the vascular channel are usually more or less symmetrical. In syphilitic endarteritis obliterans, the thickened intima and the organised thrombus in the interior of the vessel contain new blood channels; hence they do not undergo fatty degeneration, and are not usually the seat of calcareous deposits, which are so characteristic of atheroma.

Mr Pearce Gould seems to doubt whether the syphilitic and non-syphilitic forms of obliterative arteritis can be differentiated. He says:—

These considerations, in my opinion, make it very doubtful whether obliterative arteritis is a specific disease to be sharply differentiated; I would rather regard it as a pathological effect of many causes; and I would not therefore separate it from the syphilitic disease of small arteries described by Heubner and others. The chief features of obliterative arteritis are these: (1) The disease originates in the subendothelial layers of the tunica intima of the smaller arteries. In its early stage it is marked by a small-cell infiltration which later organises into a loose and vascular connective tissue. (2) This growth narrows the lumen of the vessel, and may entirely obliterate it; more often, however, thrombosis occurs and the organisation of the clot completes the permanent occlusion of the artery. (3) The disease beginning in the smaller arteries tends to spread in a centripetal direction, and may reach even the largest arteries. The thrombosis it excites also often extends rapidly and far towards the heart, much faster and further than the changes in the vessel wall, and the clot may thus spread as far as the aorta. (4) The earliest effect of this diseased artery is pain; then follow other evidences of local ischemia, and these may pass on to gangrene. These effects vary with the extent of the vascular obstruction and

the efficiency of nature's means of compensation. (5) The disease may be very chronic, slowly progressing for years, or it may run a much more rapid course. Having reached a certain point arrest may occur, and the symptoms may gradually pass away as the unaffected vessels become more and more efficient substitutes for those that have been occluded. (6) The disease, arrested for a time, may afterwards recur in the vessels of the same limb or elsewhere, and it may attack more than one vascular area simultaneously. (7) The vascular changes are not always limited to the arteries, and endophlebitis may precede or accompany the endarteritis, and the venous obstruction then modifies the effects produced on the tissues.

Relationship to Raynaud's Disease.—The condition known under the term Raynaud's disease was first symptomatically described by a Frenchman, Maurice Raynaud, in the year 1862. Its leading features are (1) *local syncope* (dead fingers or toes), (2) *local asphyxia*, in which the affected parts become red and purple or painful; and (3) *local* or *symmetrical gangrene*. The fingers and toes are the parts which are usually affected; in some cases, the ears and nose are involved; in much rarer cases, patches of gangrene develop on other parts of the body. In some cases of Raynaud's disease, there is hæmaturia; in some cases, cutaneous eruptions, such as urticaria or erythema; in some cases, cerebral or other visceral symptoms.

It is important to note that in cases of Raynaud's disease the destruction produced by the gangrene is usually very much less marked than one would expect from the apparent extent of the gangrene at its height.

Now Raynaud's disease is believed to be due or rather I should say, the phenomena of Raynaud's disease are supposed to be partly or chiefly due to a vasomotor derangement—to vasomotor spasm.

In the condition of *local syncope* the arteries are believed to be in a state of spasm shutting off the blood-supply from the affected parts (fingers, toes, etc.) and producing blanching and coldness (dead fingers).

In the condition of *local asphyxia*, the minute arteries of the affected part are supposed to be contracted, while the capillaries and veins are dilated or engorged with blood.

In the further stage of *local* or *symmetrical gangrene*, it is supposed that the minute arteries and veins are thrombosed.

The chief lesion, then, in Raynaud's disease is supposed to be vasomotor spasm—but in some cases the arteries supplying the

affected parts (those which become gangrenous) have been found diseased. In other cases, the nerves which supply the affected parts have been found to be diseased—in a condition of neuritis; in some cases of this kind, at all events, the disease of the nerves probably depends upon disease of the minute arteries which supply them with blood (vasa nervorum).

Dry gangrene may of course be due to other causes than Raynaud's disease. Anything which obstructs the blood flow through the arteries supplying the affected part—embolism or thrombosis of the arteries, narrowing and obliteration of the arteries due to any cause, such as syphilitic endarteritis, atheroma, or non-syphilitic arteritis—may cause, or be in part the cause of, the condition. The remarkable condition which is known as obliterative arteritis or Friedlander's disease is sometimes, but not always, due to syphilis. Sir Thomas Barlow has suggested, that long-continued arterial spasm, such as is present in Raynaud's disease, may ultimately perhaps give rise to obliterative arteritis.[1] In diabetes mellitus, dry gangrene is occasionally developed. In cases of this kind, both the nerves and arteries may be affected. Some twenty years ago [2] I published, with numerous illustrations, a case in which a perforating ulcer of the foot developed in the course of diabetes mellitus. In that case, the posterior tibial nerve presented marked changes, and the posterior tibial artery was atheromatous and narrowed. The affection of the posterior tibial nerve was probably, I think, due to the disease of the artery; the atheromatous posterior tibial artery and the posterior tibial nerve were bound together by dense fibrous adhesions. It is probable that in many cases of senile gangrene of the foot the same mechanism of production is present. In some cases of tabes, perforating ulcers with trophic changes in the toes with gangrene are developed, and are due to nerve changes.

Now in the first case which I will presently describe, there is not only intermittent claudication due to obliterative arteritis affecting the large as well as the small vessels, but there are also well-marked symptoms characteristic of Raynaud's disease—*local syncope* and *local asphyxia* of the fingers. As I have already stated, in some cases of Raynaud's disease the arteries going to the affected part—the tibial arteries, for example, when the toes have been gangrenous—

[1] Clifford Allbutt's *System of Medicine*, vol. vi. p. 604.

[2] *International Medical Journal*, 1888, p. 579; I have republished the case in a recent number of my *Clinical Studies* (vol. v. p. 279).

have been found to be diseased or obliterated. But so far as I know, absence of pulsation in all the arteries of the affected limb from Poupart's ligament downwards in this case has not been observed in ordinary cases of Raynaud's disease. The combination of intermittent claudication of hysterative endarteritis of the large arteries of the limb (femoral, etc.) and symptoms characteristic of Raynaud's disease is of great interest. It seems to corroborate the view that, in some cases of intermittent claudication and of obliterative arteritis, vasomotor spasm plays a part in the production of the condition, and seems to bring intermittent claudication, obliterative arteritis and Raynaud's disease into relationship. It perhaps also gives some support to Sir Thomas Barlow's suggestion that obliterative arteritis is, in some cases, due to long-continued arterial spasm.

Treatment.—The objects of treatment in cases of intermittent claudication are—

(1) *To remove the arterial condition on which the defective blood-supply depends.* In syphilitic cases the prognosis is consequently much more hopeful than in non-syphilitic cases. In obliterative arteritis which is not syphilitic and in atheromatous cases very little can, so far as we at present know, be done to cure the disease; possibly fibrolysin may prove to be of some benefit.

(2) *To keep the patient at rest and so prevent the temporary ischæmia which is the immediate cause of the symptoms.* In walking, the patient should never exceed his time limit; if any symptoms suggestive of a paroxysm (weakness, pain, etc.) develop, he should at once stop and rest.

(3) *To prevent vasomotor spasm.* The same remedies which are employed in cases of Raynaud's disease, such as the constant electric current, warm but not hot baths, shampooing, Swedish exercises, iodide of potassium, arsenic, nitroglycerine etc., should be tried. Exposure to cold and all causes likely to reflexly excite vasomotor action (contraction of the arteries) should be carefully avoided; the patient should, therefore if possible, live in a warm equable climate.

(4) *To relieve symptoms.* In the later stages of the case, i.e., when gangrene develops, the pain is usually very severe, and requires large doses of morphia for its relief.

If gangrene develops, the affected part should be covered with antiseptic wool, morphia must be given for the relief of the pain, and amputation performed as soon as the surgeon thinks it necessary

to operate. Contrary to what one might perhaps *a priori* expect, surgical interference does not seem to be contra-indicated, even when the main vessel of the limb (femoral) is completely obstructed. Two of the cases which I will presently describe, show that, even when there is no pulsation in the femoral artery, the stump may heal most satisfactorily by the first intention.

With regard to the surgical treatment of cases in which gangrene has developed, Mr Pearce Gould [1] states:—"Small mummified areas may be left to separate naturally, especially in the hand. For more extensive gangrene, which is always, I believe, of the 'mixed' form, amputation is necessary; the relief to pain that the operation affords always reconciles the patient to the mutilation. The operation should be done high above the gangrenous spot, and at a place and by a method that throws least strain upon the vessels of the part. Thus amputation through the lower part of the thigh is to be preferred to disarticulation at the knee, and musculo-cutaneous flaps are to be chosen in preference to skin flaps."

Let me now briefly describe the clinical features of some cases of intermittent claudication and of obliterative arteritis due to syphilis.

Case I. Intermittent Claudication; Gangrene of Right Foot; Obliterative Arteritis of the Right Leg; Amputation, etc. G. S., aged 46, married, ship's steward, was admitted to the Edinburgh Royal Infirmary on February 14th, 1907, suffering from pain in the right foot and leg.

History prior to the Onset of the Present Illness.—For five years before his first admission to the Infirmary (February 1907), he had acted as a steward on board a steamer trading between Liverpool, Glasgow and Montreal; before that, he was for twenty years in the Army. Up till eighteen months before his first admission to the Infirmary (October 1905), he enjoyed excellent health. When in the Army he was an athlete (a runner, swimmer, etc.). He is not flat-footed.

He emphatically states that he has never had venereal disease, either syphilis or gonorrhœa. I may further say that there are no signs of previous syphilis, either congenital or acquired, on the surface of the body.

The patient states that he has always been a moderate smoker—at the most 2½ ozs. a week, and that he has been steady as regards alcohol; for the past five years he has been a teetotaller, before that he was a moderate drinker. His surroundings have been satisfactory. He has never been much exposed to cold; as a ship's

[1] *Lancet*, March 15, 1902, p. 722.

steward he was almost always under cover. These are important facts from an etiological point of view, for some authorities have suggested that intermittent claudication and the form of endarteritis obliterans from which this patient suffers are in some cases due to excessive smoking, exposure to cold and alcoholic excess.

FAMILY HISTORY.—This is very good. His father died at the age of ninety-eight, and his mother at the age of eighty-nine, from old age. His two brothers were accidentally killed. His two sisters are alive and well.

He has three healthy children; two other children died in infancy—one of pneumonia. His wife has had no miscarriages.

HISTORY OF THE PRESENT ILLNESS.—The patient states that he was quite well till September 1905. He then had an attack of influenza. In October 1905, he began to suffer from what he terms sciatica in the right leg—pains in the right leg, shooting from the hip to the ankle. Shortly after these pains developed, he noticed that walking for any distance caused pain, stiffness and cramp in the right leg, and that, if he persisted and tried to walk off the stiffness, the leg became extremely rigid and the tendons stood out as the parts were being forcibly drawn asunder. So long as he was at rest there was, he says, no pain, weakness or stiffness in the leg, but walking a short distance—300 yards—would bring on these symptoms and make him limp. After resting for a short time (half an hour or so), the pain, stiffness and weakness passed off. He says that when the pain and weakness came on in the leg, the veins used to look as if they were full of blood, the leg, more especially the foot, became dark in colour, and little red spots were apt to develop on the surface of the skin.

Another condition which developed about the same time as the intermittent claudication was "*local syncope*" followed by "*local asphyxia*" in the middle and ring fingers of the right hand. These fingers used to become pale and dead (*local syncope*) after the pallor had lasted for a certain time (sometimes an hour), the affected parts became red, purple and very painful (*local asphyxia*). The pain was of a stinging, burning character. Before the pain and weakness in the right leg (intermittent claudication) developed he never suffered from dead fingers. The dead condition of the fingers used to develop very frequently, usually several times a day.

Some little time after the intermittent claudication, local syncope and local asphyxia developed, small red spots, about the size of a threepenny piece began to appear on the right (affected) leg—these spots, the patient says, were very painful to the touch, they were only very slightly if at all raised above the surface of the skin. They developed in successive crops, on the outer side of the thigh from the hip to the knee, and on the inner side of the leg from the knee to the ankle—this distribution is peculiar and difficult to explain. The development of the spots was preceded by a feeling of

Fig. 1.—Foot of the patient described in the text showing gangrene.

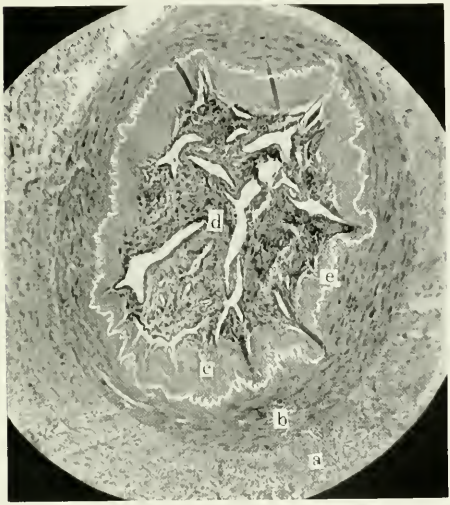

Fig. 2.—Microphotograph (touched up to bring out the elastic lamina and middle coat) of a section through the upper part of the posterior tibial artery in the case of arteritis obliterans described in the text.

The letter *a* is placed on the outer coat; *b*, on the middle coat; *c*, on the thickened inner coat; *d*, on the organised thrombus in the centre of the vessel; and *e*, on the inner elastic lamina which separates the inner and middle coats. A number of empty spaces (new vascular channels) are seen in the organised thrombus.

PLATE XIV.—TO ILLUSTRATE DR BYROM BRAMWELL'S ARTICLE.

pain and stinging in the skin. Each crop of spots would last two or three days and then disappear; before they faded, the spots became darker—more dusky—in colour. A new crop of spots would then develop in the course of three or four days. These painful spots continued to develop for several months.

Two weeks before the patient's first admission to hospital he began to suffer from severe pain and weakness in the right big toe and pain in the right foot and leg; after a few days, the toe became red and swollen and its tip black (gangrenous).

Condition on the Patient's first admission to the Infirmary (*February* 14, 1907).—When admitted to hospital the patient was found to be a spare man; his height was 5 ft. 8 ins., and his weight (in dressing-gown) 8 st. 8½ lbs.—in health he used to weigh (in clothes) 10 st. 13 lbs.—he had probably therefore lost at least a stone in weight since the illness commenced.

The temperature was 97°; the pulse 98 and the respirations 20 per minute.

The big toe of the right foot was swollen and red, the tip black and gangrenous. The patient complained of intense burning pain in the toe; also of a stinging pain in the right foot and leg. Covering the toe with cotton wool increased the pain; it had therefore to be kept uncovered, but protected by a cage. The pain in the toe was so intense that morphia had to be given at frequent intervals; trinitrine was tried, but it gave no relief.

The sensibility of the skin of the right leg, and in fact of all parts of the body, to touch, pain, heat and cold was quite normal. The *reflexes* (superficial, deep and organic) were all normal. The *pupils* were equal and active. The *fundi oculi* were normal. There was *no paralysis*.

The *urine* was normal; there had never been any hæmatinuria. The *heart* was normal.

The *radial pulses* were of good volume and strength; their walls were somewhat, but not much, thickened. The *temporal arteries* were prominent and their walls seemed somewhat thickened.

No pulsation could be detected in the arteries of the right leg below Poupart's ligament—this observation was confirmed by Mr Cathcart, who kindly examined the patient. *Pulsation could be felt, but it was much less forcible than normal, in the femoral, popliteal, and anterior pedal arteries of the left leg. No pulsation could be felt in the posterior tibial artery of the left leg.*

After the patient's admission to hospital in February 1907, the gangrene gradually but slowly extended, and ultimately involved the greater part of the right foot (see Fig. 1), and the patient continued to suffer from very severe pain in the right foot and leg; he had to be kept continuously under large doses of morphia for its relief.

On May 31, 1907, the right leg was amputated by Mr Cathcart

below the knee; the operation was performed under chloroform. No tourniquet or compression of any sort was applied. There was practically no bleeding at the operation. All of the large arteries were obliterated and thrombosed. There was a little venous oozing; some of the veins and a few small arteries in the muscles were ligatured; the large arteries of the leg were also ligatured as a matter of precaution.

The patient made a rapid recovery and was discharged from hospital a fortnight after the operation, the wound healed by first intention, the pain completely disappeared after the operation.

The arteries of the amputated limb were examined microscopically, their condition is represented in Figs. 2, 3 and 4.

In the section reproduced in Fig. 2 there is great thickening of the intima, and an organised thrombus, in which numerous new vascular channels lined by endothelium are present, completely fills the interior of the constricted vessel. The middle and outer coats are, practically speaking, normal.

In the section reproduced in Fig. 3 a well-marked atheromatous patch is present, outside the inner elastic lamina, which separates the inner and middle coats. This atheromatous patch is of importance I think, for it seems to add confirmation to the view that the arterial disease is not syphilitic.

In some of the smaller vessels (see Fig. 4) the middle coat is thickened.

Subsequent Progress of the Case.—The patient continued well for six months—until the beginning of the present year, he then began to suffer from a recurrence of the same symptoms—pain and disturbance of the circulation with blackness of the toes, but no gangrene, in the left leg.

Readmission to Hospital.—The patient was readmitted to the Infirmary, on April 23, 1908, looking pinched and ill. His nose was blue; he complained of severe tingling and burning pains in the left foot and leg. He stated that the toes of the left foot became at times blue and discoloured, there was no gangrene.

No pulsation could be felt in the anterior pedal and posterior tibial arteries of the left foot, nor in the left popliteal artery. A very feeble pulsation was felt in the left femoral artery.

The radial pulses were unequal in size, the left being considerably larger and fuller than the right (see Fig. 5). Both radial arteries, more especially the right, were thickened. The coats of the right radial artery seemed much more thickened than they were a year ago.

The blood pressure in the upper extremities was equal = 115 mm. of mercury. On applying the blood-pressure apparatus to the left calf, very feeble flickering of the mercury was observed, whether this was due to feeble circulation in the main vessels of the leg or to

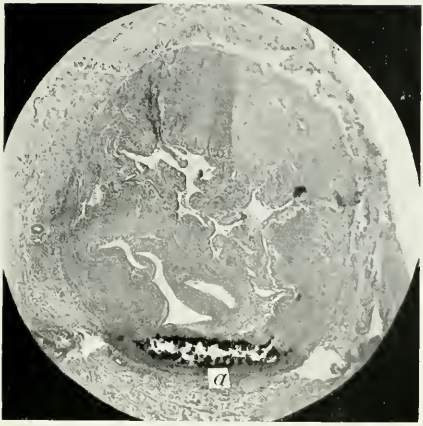

Fig. 3.—Microphotograph of a section through the posterior tibial artery in the case of obliterative arteritis described in the text.

The letter *a* is placed just outside the calcareous patch described in the text.

Fig. 4.—Microphotograph of a section through the lower part of the posterior tibial artery in the case of obliterative arteritis described in the text, showing thickening of the middle coat.

The letter *a* is placed on the outer, *b* on the middle, and *c* on the inner coat.

Plate XV.—To Illustrate Dr Byrom Bramwell's Article.

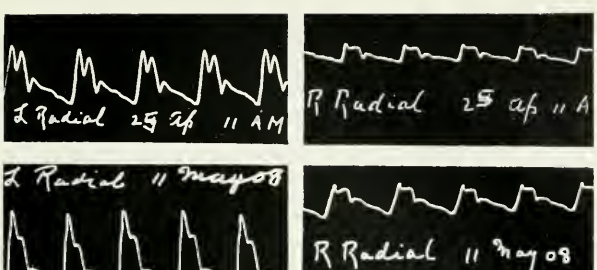

FIG. 5.—Sphygmograms of the radial pulses, taken on April 25th and May 11th, 1908, in the case of arteritis obliterans described in the text.

When the sphygmogram was taken on April 25th, the left hand was hot, painful, and throbbing, but presented no definite changes in colour; the right hand was cold and bluish in colour, but not painful.

When the sphygmogram was taken on May 11th, the maximum blood pressure was 140 mm. of mercury in each arm, but in the case of the right arm there were regular periods in which the pressure was about 10 mm. of mercury lower.

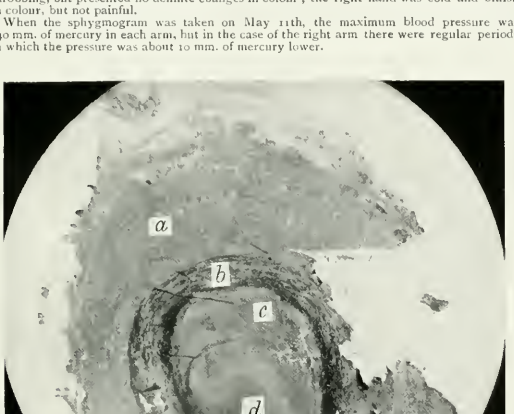

FIG. 6.—Photograph of a section of the left common carotid artery in the case of syphilitic arteritis described in the text. The channel is completely obstructed.

The letter *a* is placed on the outer, *b* on the middle, and *c* on the inner coat, the letter *d* on the organised tissue blocking the lumen of the vessel; part of the greatly thickened outer coat has become detached in the process of mounting. (See Dr Shennan's description of the microscopical appearances.)

PLATE XVI.—TO ILLUSTRATE DR BYROM BRAMWELL'S ARTICLE.

pulsation in the small anastomotic vessels could not of course be determined.

The *heart* and *urine* were normal. There were no alterations of *voluntary motion*, *sensation*, or *reflex action*.

The red corpuscles numbered 6,900,000, and the white corpuscles 8000 per cubic millimetre; the hæmoglobin equalled 100 per cent. (On May 21, the red corpuscles numbered 6,700,000 per cubic millimetre, and the hæmoglobin equalled 100 per cent.)

The patient stated that for a month prior to his readmission to hospital the ring and middle fingers of the *left* hand had frequently become dead and then blue. The *local syncope* and *local asphyxia* were, it will be remembered, previously limited to the middle and ring fingers of the *right* hand; they now affect the left as well as the right hand. Since the patient's readmission to hospital, several attacks of local syncope and local asphyxia have occurred; all the fingers of the right hand and the middle and ring fingers of the left hand are now affected.

Prognosis.—Now I greatly fear that in this poor man's case the obliterative arterial lesion will continue to develop, and that his left foot will in the course of time become gangrenous and will have to be amputated. Further, I fear that in the course of time the upper limbs will probably also become affected—indeed, the occurrence of local syncope and local asphyxia in the fingers of both hands and the condition of the right radial artery (its coats are much more thickened than they were a year ago) seem to show that the vessels of the upper extremities are already involved. But in this respect the case is not peculiar, for cases have been described in which gangrene has successively developed in two or even three limbs, and successive amputations of two or even three limbs in the same patient have had to be performed as a result of this remarkable obliterative endarteritis.

Case II. Obliterative Arteritis of the Femoral and other Arteries of both Legs; Gangrene of the Right Foot; Amputation; Healing by First Intention, etc. Isaac G., aged thirty-six, widower, Jew, traveller for jewellery, was admitted to the Edinburgh Royal Infirmary on April 14, 1902, suffering from pain in the right foot and gangrene of the right big toe.

Previous History.—The patient states that he has never had syphilis or any form of venereal disease. He is a teetotaller. He has always been a heavy smoker; when well, he used to smoke half an ounce of tobacco (cigarettes) per diem. He has taken tea to excess. He has always been a nervous man.

He enjoyed good health until four years ago, when (May 1898) he began to suffer from pain in the legs after walking, tenderness in the middle of the right thigh, dyspepsia and chronic diarrhœa. He was treated as an out-patient and improved.

He states that during the past five years, after taking opening

purgative medicine, he was apt, when lying quiescent in bed at night, to be troubled with violent spasmodic jerking movements of the head and legs; the head would be violently jerked backwards and the legs flexed on the abdomen.

He continued to follow his occupation, that of a travelling jeweller, until the present illness commenced, some ten weeks ago. His occupation involved a good deal of walking (three, four, and sometimes even ten miles a day).

Ten weeks ago he noticed that his right foot and leg became numb, more especially during the night, and, at times, dead; the foot and leg as far as the knee were pale and blanched during these attacks. When he walked the ankle felt stiff and he dragged the foot.

A week later the big toe and foot became swollen and red and very painful; "the foot and leg felt," he says, "as if it were too full of blood and as if it were going to burst." A doctor whom he consulted diagnosed gout. He was admitted to Chalmers Hospital, where he remained for three weeks. While in Chalmers Hospital, he had some sort of fit during the night—he does not think that he quite lost consciousness; he felt, he says, as if the blood were leaving his arms and legs and rushing to his body; his jaws were clenched and chattering; he broke out into a cold, clammy sweat; the room seemed revolving round him; his legs and arms trembled violently; he had to be held down, he says, by two nurses, who gave him brandy.

Four weeks ago, a blister formed at the end of the big toe. *A fortnight ago*, the toe became black and discoloured (gangrenous).

Since his illness commenced he has lost 1 st. 9 lbs. in weight, and has become very weak. The pain in the foot and leg has been very great.

Family History.—His father, mother, and one sister are alive and well; he did not know their ages. Seven brothers and sisters died when young; he did not know their ages at death or the causes of their deaths.

Condition on Admission.—The patient was a thin, non-muscular, nervous-looking man. There were no stigmata of syphilis, either congenital or acquired. The big toe of the right foot was gangrenous. He complained of intense pain in the right foot and right leg.

The temperature was 98·4°, the pulse 96, the respirations 22.

The *radial pulse* was of good volume and strength; the coats of the artery were slightly thickened. The *heart* was normal. The *urine* was normal. There was no *paralysis*. The *sensibility of the skin* to touch, pain, heat, and cold was normal in the right foot, leg, and other parts of the body. The *reflexes* (superficial, deep, and organic) were normal. The *pupils* and *fundi* were normal.

No pulsation could be felt in the arteries of either leg below Poupart's ligament.

Treatment and Results.—The affected toe was covered with aseptic wool; nitroglycerine was given internally, and the pain relieved by frequent hypodermic injections of morphia.

The gangrene slowly but gradually extended, and ultimately involved a considerable portion of the right foot. The pain continued to be very severe.

Amputation.—*On May* 9, Mr Cathcart amputated the leg through the knee-joint. No tourniquet or other form of compression was applied. The operation was practically bloodless; only one small muscular branch bled. The femoral artery was completely occluded, but was ligatured as a matter of precaution.

(On subsequent examination, the femoral artery was found to be occluded for half an inch below the seat of amputation; below this point it was patent; in the middle of the leg, the anterior and posterior tibial arteries were completely obliterated; the dorsalis pedes was patent.)

The wound healed by the first intention, and the patient was quite free from pain after the operation.

Intermittent claudication occasionally occurs without any obvious defect of the arterial circulation in the affected limb. If this is so, one must suppose that, in such cases, vasomotor spasm is the cause of the condition. The following are the notes of a case which I recently met with in which there was no defect in the arterial circulation. On the first occasion on which I saw the patient I thought the arterial circulation was distinctly defective in the lower part of the femorals, in the popliteals, and in the anterior pedal and posterior tibial arteries on both sides. Mr Cathcart, who kindly saw the case, could not satisfy himself that there was anything abnormal. Curiously enough, I have to-day had the opportunity of again seeing the case. To-day, the pulsation in the arteries of both legs (femorals, popliteals, posterior tibials, and anterior pedals) is perfectly good and strong.

Case III. Typical Intermittent Claudication affecting both Legs in a Very Muscular Man; ? No Obvious Defect of Arterial Circulation. A. B., aged thirty-three, single, medical man, was seen on April 18, 1908, complaining of typical and very marked attacks of intermittent claudication.

The patient was a very muscular, healthy man, a teetotaller and very moderate smoker (three cigarettes per diem); he had never had syphilis nor any other venereal disease. For the past winter he had walked little and had done most of his work on a motor bicycle.

During the past winter he had suffered from intermittent claudication. He gave the following account of the attacks:—"After

walking a short distance I feel pain in the thighs and calves, numbness and tingling in the legs, a feeling of heat in the legs, cramp and loss of power in the legs. These symptoms are so marked that I have to stop; I cannot go on walking or I would fall.

"After resting for a short time (five or ten minutes) the symptoms pass off and I feel all right again.

"I have never noticed any alteration in the colour of the legs."

The patient was, as I have said, a very muscular man. All the organs appeared to be absolutely normal. No derangement of motion, sensation, or reflex action could be detected in the legs or elsewhere.

The pulsation in the upper part of the femoral arteries was strong, but in the vessels of both legs below the middle of the thigh, where the artery is lost in muscle, difficult to feel, pulsation could be felt but with some difficulty in the popliteal, anterior pedal, and posterior tibial arteries. Mr Cathcart could not satisfy himself that there was any abnormality of the circulation. He thought the difficulty in feeling the arterial pulsation in the lower part of the femorals, in the popliteals, and in the anterior pedal and posterior tibial arteries due to the great muscularity of the patient.

On July 1st, 1908, the arterial pulsation in both legs was perfectly normal. The posterior tibial and anterior pedal arteries could be felt pulsating strongly.

A few years ago I saw a patient suffering from tabes in whom no pulsation could be felt in either femoral artery. This obliteration of the femoral arteries had certainly been present for five years before the tabetic symptoms developed. Prior to the tabes, the patient had merely complained of weakness in the legs. When I saw the patient in the year 1902, I was not familiar with intermittent claudication, and made no inquiry as to whether the difficulty in walking was intermittent and attended with pain, spasm, numbness etc. The notes are as follows:—

Case IV. Tabes and Absence of Pulsation in both Femoral Arteries, probably due to Syphilitic Endarteritis Obliterans. Male, aged forty-four, married, seen on January 27, 1902, suffering from typical tabes and absence of pulsation in both femoral arteries.

The patient absolutely denied syphilis and gonorrhœa.

The tabetic symptoms were first noticed a year before I saw him (in January 1901), and were highly characteristic, viz.—ataxia in the legs, a characteristic tabetic gait, Rombergism, some ataxia in the arms, severe lightning pains in the legs, arms, and trunk, pain in the lower part of the back, girdle sensation round the lower part of the abdomen, numbness in the arms and legs, coldness of

the feet; muscular analgesia in calves; hypotonia; knee-jerks and Achillis-jerks absent; plantar reflex flexion; abdominal reflex absent; urination—dribbling; bowels—difficulty in getting a complete evacuation, has to go several times a day; diminution in sexual desire and power.

Absence of Pulsation in the Femoral Arteries.—Six years before the tabetic symptoms developed (in the year 1895), the patient began to complain of weakness in the legs.

Five years before the tabetic symptoms developed (in the year 1896), he consulted Mr Jonathan Hutchinson, who found that there was no pulsation in the femoral arteries.

When he came under my notice (in the year 1902) the same condition—complete absence of pulsation in both femorals and the other arteries of both lower extremities—was present.

The Diagnosis was: Tabes and obliterative endarteritis, probably affecting either the lower part of the abdominal aorta or the great iliac vessels.

Notwithstanding the patient's denial of syphilis, the arterial condition was thought to be in all probability syphilitic. This opinion was based on the facts that obliterative endarteritis is often, and tabes almost invariably (? invariably) syphilitic in origin. Why the patient consulted Mr Jonathan Hutchinson, who is a great authority on syphilis, I do not know; but it is perhaps an additional argument in favour of the syphilitic nature of the condition.

I have already stated that complete obstruction of the femoral arteries does not necessarily produce intermittent claudication. The following are two cases in point. In the first case, the lower end of the aorta, the iliac and femoral arteries were completely occluded by a dense organised thrombus. Up to the time of his death, the patient led an active life and made no complaint of weakness, pain, or numbness in the legs. He also was the subject of the rare condition—a true cardiac aneurism, and yet (with the exception of a severe attack of angina pectoris seven years before his death), had not suffered from any cardiac symptoms.

Case V. Case of Thrombosis of the Lower End of the Aorta and Great Iliac Vessels; No Symptoms of Intermittent Claudication; Cardiac Aneurism. A. B., a gouty man, aged forty-nine, was seen in consultation several years ago. For nine months previously, he had suffered from attacks of diarrhœa and recurring abdominal pain, sometimes very severe, about the umbilicus. Of late, he had been losing flesh, but had led an active life (riding and hunting), and had had no symptoms suggestive of intermittent claudication.

Seven years previously he had had a severe attack of angina

pectoris, which was thought to be due to an aortic aneurism; since this attack he had never been troubled with his heart.

I saw the patient on a Wednesday. Two days before I saw him (on Monday) he had been out all day hunting; it was a very cold day. On Monday evening, he complained of severe pain in the region of the umbilicus, and of a feeling of distension and flatulence; the pain was intermittent and did not radiate in any direction; there was no tenderness on pressure.

On examination, I could detect nothing in the abdomen; everything appeared to be normal. A well-marked systolic murmur was audible at the base of the heart, but so far as I could see there was no evidence of aneurism. The patient was said to be gouty.

I thought the condition was one of persistent and recurring colic, and gave a favourable prognosis. The pain was not relieved by morphia; seven doses of 15 minims of the liquor.

Thirty-six hours after my visit, the patient went to stool, and a few minutes later was found dead in the w.c.

Post-mortem examination showed:—(1) a cardiac aneurism—a localised dilatation of the wall of the left ventricle, about the size of a hen's egg, filled with firm laminated clot; the wall of the left ventricle at the seat of the aneurism was converted into fibrous tissue and was in places gritty and calcareous.

(2) Slight atheroma of the aortic arch.

(3) No affection of the aortic or other cardiac valves.

(4) Narrowing of the lower part of the abdominal aorta, and complete obstruction of the lower part of the abdominal aorta, iliac and femoral arteries, with firm organised clot.

On inquiry I subsequently learned that the patient had been riding and hunting daily until three days before his death; that he had not complained of numbness, pain, weakness, or loss of power in the legs, but his lower extremities had, to the touch, felt much colder than his arms.

The absence of both sensory and motor symptoms in the lower extremities in this case, in which the lower end of the aorta, the iliac and femoral arteries were completely occluded, is remarkable and can only, I think, be accounted for by supposing that the condition was very slowly and gradually developed, and that the musculatory circulation was unusually well established. The severe abdominal pain and recurring attacks of diarrhœa are interesting. In one of the other cases which I have related (Case II.), in which the circulation through both femorals was obstructed, there was also abdominal pain and chronic diarrhœa. Whether in that case the lower end of the aorta was obstructed or not I am unable to say.

Case VI. Gangrene of the Second Toe of the Right Foot.

AND BLUENESS OF THE TOES OF THE LEFT FOOT; COMPLETE ABSENCE OF PULSATION IN THE ARTERIES OF BOTH LEGS; NO INTERMITTENT CLAUDICATION. M. O'B., aged 66, a pitman by occupation, was admitted to the Edinburgh Royal Infirmary on 16th June 1908, complaining of pain and swelling of the right foot and a small ulcer on the dorsum of the right foot.

Seven weeks before admission to hospital he began to complain of pain in the left foot and a blister under the toes. A few days later the right foot became affected. Up to the day before his admission to hospital he had continued regularly at work.

On admission to hospital, the second toe of the right foot was blanched, the foot was red and swollen, a small ulcer about the size of a pea was present on the dorsum; no pulsation could be felt in either femoral arteries below Poupart's ligament or in any of the arteries of either leg. The affected toe gradually became blue, and at the present time (July 1st, 1908) is gangrenous. The toes of the left foot also look blue. It seemed probable that gangrene in both feet would develop, and that amputation of both legs would ultimately be required.

Another remarkable case in which the left common carotid artery was completely, the right common carotid artery almost completely, and the left subclavian and right subclavian arteries considerably obstructed by syphilitic endarteritis, occurred recently in my hospital practice. In this case, notwithstanding the very defective blood supply to the brain which must have been present, there were no head symptoms until twelve hours before death, when the patient became delirious. Further, it is remarkable that notwithstanding the presence of a gumma which involved the brain, its membranes, and the parietal bone, the patient never made any complaint of headache. I have not time to give you full details regarding it, but the following are the leading features of the case:—

CASE VII. SYPHILITIC DISEASE OF THE AORTA, PULMONARY ARTERY, AND GREAT VESSELS ARISING FROM THE AORTIC ARCH; COMPLETE OCCLUSION OF THE LEFT COMMON CAROTID; ALMOST COMPLETE OCCLUSION OF THE RIGHT COMMON CAROTID; GREAT NARROWING OF THE RIGHT AND LEFT SUBCLAVIAN ARTERIES; NO HEAD SYMPTOMS; INTRACRANIAL GUMMA. A. K., aged thirty-six, married, an upholsterer, was admitted to the Edinburgh Royal Infirmary on October 12, 1907, complaining of shortness of breath, pain in the upper part of the chest and root of the neck, and indigestion.

The patient walked up to the Infirmary. On admission he was seen and examined by Dr Bramwell. He looked very pale and ill. The condition of the heart, etc., is described later.

History ascertained partly from the patient himself, and partly from his wife, an intelligent woman, after his death). During the past summer his wife noticed that he was short of breath on going up a hill, but he made no complaint of illness. At the end of July, he walked from Kinghorn to Burntisland, a distance of three miles, and was quite fresh afterwards. After this, the shortness of breath became more marked, he looked at times ill, and lost flesh, but he continued to work until six weeks before his admission to hospital. He then (six weeks before admission to hospital) took to bed. His breathing became very difficult; he could not lie down because of the shortness of breath; he scarcely slept at all during these six weeks; his feet swelled; he complained of pain at the bottom of the neck, worse on the left side, and of flatulence and indigestion. The pain in the neck was worse at night. He never, his wife says, complained of headache, vomiting, or giddiness; he was quite bright mentally.

The patient denied syphilis; he had been married for ten years, but had had no children; there had been no miscarriages. He was practically a teetotaller, and a moderate smoker (2 ounces per week).

Condition on Admission.—The patient looked very ill; he was pale and very short of breath; the feet and legs were slightly swollen. The temperature was 97°, the pulse 112, and the respirations 32. He was thin (height 5 ft. 9½ ins.; weight 8 st. 7¾ lbs.). On the right leg and thigh there were scars of serpiginous ulcers and old rupia; there was also a scar on the glans penis, and scars in the groin.

The heart was enlarged, the apex beat was in the 6th interspace well outside the nipple line, and 5 inches from the mid sternum. A double aortic and a systolic mitral murmur were present. The pulse was 112, very small in volume, and of low tension; the radial coats were not thickened; the two radial pulses seemed of equal size.

Over the upper part of the manubrium sterni there was some bulging and marked dulness; the patient complained of pain in this situation and at the root of the neck; there was well-marked pulsation in the suprasternal notch.

The *urine* contained a small quantity of albumen. The *tongue* was furred, the *breath* foul, the *liver* considerably enlarged. The *pupils* were equal and reacted normally; the *knee-jerks* were sluggish but equal. There was no *paralysis*.

The patient made no complaint of headache or giddiness; mentally he seemed quite bright.

Progress and Result.—Notwithstanding treatment the patient rapidly got worse and died, on *October* 10, at 4.30 A.M. During his stay in hospital he complained of pain in the chest, was very sleepless, and for the last twelve hours was very delirious.

Post-Mortem Examination.—This was made by Dr. Shennan

on October 18. Dr Shennan's report is as follows (I omit many particulars regarding the organs) :—

"The anterior border of the right lobe of the liver reached to the level of the umbilicus. The right pleural cavity contained 25 and the left 12 ounces of blood-stained fluid.

"The heart was considerably enlarged. There was evidence of old pericarditis at the base and surrounding the roots of the great vessels.

"*Pulmonary Artery.*—The cusps were thickened and elongated. On the inner surface of the artery, in its right posterior part adjacent to the aorta, and immediately above the pulmonary valve, there was a localised thickening which measured about 1 inch (2.5 cm.) in diameter; a considerable proportion of this thickening consisted of firm, yellowish, necrotic material almost certainly of gummatous nature; the intima over the thickened area showed patchy congestion.

"The *aortic orifice* was dilated and incompetent; the valvular segments thickened and elongated.

"The *aorta* was dilated in fusiform fashion down to the end of the transverse part. The inner surface was irregular, atheromatous, pitted in parts with atheromatous ulcers and in parts covered with a delicate deposit of lymph. About the middle of the ascending part of the aorta there was a saccular aneurism about the size of a hazel-nut passing backwards and to the right.

"The walls of the aorta throughout, but particularly at the origin of the great vessels, were infiltrated with yellowish tough necrotic material, evidently of gummatous nature; it measured in parts $\frac{1}{4}$ inch (0.6 to 0.7 cm.) in thickness.

"The *innominate artery* was reduced to size of a goose-quill near its origin (*i.e.* internal diameter).

"The *right subclavian* was pervious but greatly constricted, to about $\frac{1}{20}$th of an inch in diameter, at its commencement (1.5 to 2 mm.), then it abruptly expanded to form a vessel practically of normal size.

"The *right common carotid*, the lumen of which was fairly large at the point where the microscopic section was made, became, lower down, extremely constricted, and for a short distance—about $\frac{1}{2}$ an inch (1 cm.)—after leaving the innominate artery, was practically impervious to the finest probe. One inch above the aorta, the free channel in the right common carotid was about 2 mm. in diameter. Its branches were practically normal.

"The *left common carotid* was completely blocked up to a point just below the origin of the superior thyroid, etc.

"The change in the *left subclavian* was very similar to that in the right subclavian. The constriction of the lumen was not, however, quite so extreme.

"*Head.*—On the right side, about the middle of the parietal bone,

there was an area in which the inner table was thin and softened. This corresponded to an area in which the dura was adherent and infiltrated with yellowish gummatous material. This again was adherent to brain substance, the gummatous change involving the arachno-pia mater and also extending into the cortex for a short distance—nearly 1 cm. at the centre of the area. The adjacent part of the inner table showed the 'caries sicca,' typical of tertiary syphilis. The arachnoid was thickened, and the fluid in the space was increased.

'The central part of the meningeal gummatous change corresponded to the superior parietal lobule, and it extended forwards for a short distance on to the ascending parietal convolution, and also backwards. The supra-marginal gyrus had escaped.

"MICROSCOPICAL APPEARANCES.—*Left Common Carotid* (see Fig. 6).—The lumen is completely blocked. In the centre there is fibro-cellular tissue undergoing necrosis. The fibrils are hyaline, and between them are numerous particles of chromatin derived from the nuclei of degenerated cells. Next to this is a layer of very cellular vascular tissue, which blends intimately with the wall of the vessel. In this there are occasional giant-cells. A little farther out the concentric lines of wavy elastic tissue, brought out best in the sections stained with orcein, indicate the position of the remains of media. This coat is very much broken up, showing numerous patches of cellular connective tissue in relation to vessels which are penetrating the wall from the outer coat. Many of the larger vessels in the media, as well as most of those in the adventitia, have thick proliferated walls.

"The adventitia is very thick from new formation of dense, partly hyaline, fibrous tissue.

"*Right common carotid* (see Fig. 7).—Section made about one inch and a half from the aorta.—The changes here correspond closely to those seen on the left side. The lumen, however, is not completely blocked, it is lined by a thick layer of hyaline fibrous tissue, containing some small bloodvessels, which resembled the tissue blocking the lumen of the left common carotid.

"Outside this there is a vascular cellular connective tissue layer, parts of which are undergoing early irregular cascation. It contains occasional arrangements of epithelioid elements, resembling, but larger than, the ordinary grey tubercle, and containing more fibrous tissue than it usually does.

"The media is not quite so much broken up, and parts of it can readily be distinguished even with ordinary stains.

'The adventitia corresponds in appearance to that on the other side, except that the vasa vasorum have thicker walls and are more typically syphilitic.

"*Pulmonary Artery*.—The necrotic part of the thickening is typically gummatous; the remainder of the thickening is composed of cellular, very vascular connective tissue, which surrounds the

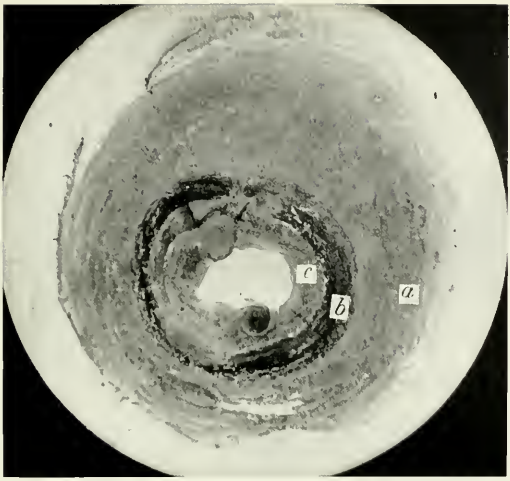

FIG. 7.—Photograph of a section of the right common carotid artery at a point 1½ inch above the aorta, showing great thickening of the inner and outer coats, etc.
The letter *a* is placed on the outer, *b* on the middle, and *c* on the inner coat.
(See Dr Shennan's description of the microscopical appearances.)

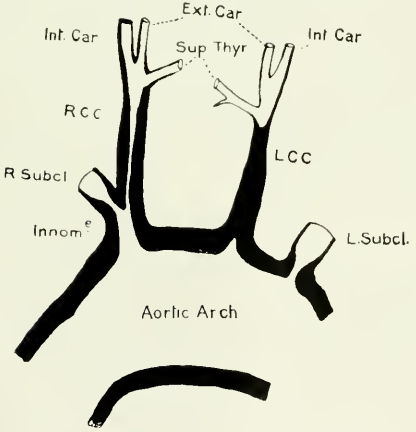

FIG. 8.—Rough diagram (drawn by Dr Shennan) showing the condition of the vessels arising from the aortic arch.

PLATE XVII.—TO ILLUSTRATE DR BYROM BRAMWELL'S ARTICLE.

caseous area and extends outwards into the intima. A remarkable feature is the great number of epithelioid cells and giant-cells which are present in this cellular tissue.

"*Inosculatory Circulation.*—As to the manner in which the circulation to the head and upper extremities was carried on, this must have passed through collaterals.

"A certain amount of blood had reached the vertebrals by way of the subclavians direct, and also by way of the inferior thyroids to the superior thyroids, and thus to the external and internal carotids. (The communication between the carotids and vertebral arteries by way of the ascending pharyngeals may also have been of some service.)

"In addition, the second intercostals (1st aortic) had supplied blood through the first intercostals to the subclavians, and thus to the vertebrals and inferior thyroids (and other collaterals).

"Lastly, an additional supply had been obtained by way of epigastric and internal mammary arteries to the first part of subclavian and so to vertebrals, etc.

"If any blood had passed through the right common carotid, the quantity had been quite negligible." (The condition of the circulation is diagrammatically represented in Fig. 8.)

3. PUBLIC MEDICINE FROM THE SOCIOLOGICAL STANDPOINT

By Alexander James, M.D., F.R.C.P. Ed., Consulting Physician, Edinburgh Royal Infirmary.

I am never tired of reiterating that famous saying of Descartes, that in our efforts to render more and more perfect the condition of humanity, it is to medicine that we must look for the means.

To all of us, the great truth in this statement must be apparent enough. It is the function of the doctor to seek to interpret the meanings of all those multitudinous and complex processes which together work towards the growth and development of individual living beings. Inasmuch as what we call societies are merely aggregates of individuals, showing like processes of growth and development and governed by the same laws as govern the individual, it is evident that the man whose life's work it is to investigate and interpret the laws which govern the life of the individual, must of necessity acquire information which will fit him specially well to investigate and interpret the laws which govern society.

At no time probably in history has the importance of the information which the doctor can give to society been more appreciated than at the present time. Even in military life, where it is the

function of one group to exterminate the other. Napoleon's famous saying that Providence always sides with the biggest battalions must be accepted now with the proviso that this is so only as long as these battalions are under efficient medical care, and the Japanese have shown this as cogently in the right way as it has been shown in the wrong way up to this time by most of the European nations.

In civil life, the importance attached by the community to the doctor's advice and guidance is now all-pervading. Whilst formerly the doctor was present and all-powerful in the sick-room and in the hospital, now he is present and active in the school, the workshop, the factory, and the mine. Whilst formerly he was called when pain and suffering were to be allayed and assuaged, now he goes or is sent to work among men and women in health and vigour, so as to ensure that no evil conditions are present which might cause that health and vigour to be impaired. Whilst formerly he was called in by the individual, and the remuneration which he received for his services depended largely on the capability of individuals individually or collectively, to pay him, now he is to a large extent employed by the community, and he is recompensed by the community, it must be said none too largely. It has to be admitted that with those changes there are some other changes for which we doctors are not altogether grateful. Whilst formerly the doctor could regard himself as the welcomed guide, counsellor, and friend of every individual in the community, now with compulsory notification and isolation, with medical inspection in schools, etc., and with compulsory birth notification thrown in, he is apt to realise as he moves about in his daily work that his appearance is not always welcomed, and that the feelings with which he is regarded by the people are somewhat akin to the feelings with which they regard the official, the detective, and the jailer.

To the individual doctor, endowed with the genuine spirit of the human healer, some of these modern developments cannot but be a matter for sincere regret. But if we are assured that these developments are leading to a higher standard of health and vigour for the community, we doctors must simply acquiesce and accept.

This leads to the question which I wish to lay before the Society to-night, viz., Is it the case that those modern developments in connection with what is really public medicine are working for the public good?

Among these developments, the view that with efficient notification and isolation the community can through its public health

officials, stamp out all the so-called specific diseases, holds a very high place, and for the last 20 or 25 years more or less thorough efforts in this direction have been put into practice all over the country.

It has been recognised that this assumption by the community of responsibilities which should reasonably fall on individuals would be very costly. Confidence, however, in the wisdom of the medical advice offered has caused communities to respond loyally to the large pecuniary calls made upon them.

It has been recognised that the assumption by communities of individual responsibilities has other disadvantages. Apart from the fact that it strikes a blow, and often a painful one, at family life, which, in its turn, is the organic basis of social life, it has the effect of lessening that feeling of responsibility in the individual of which is begotten providence, foresight, and frugality. Again it will be said that the end justifies the means, and that whatever leads to the health and welfare of the community as a whole will more than counterbalance any disadvantage to the moral or intellectual sense of the individual.

The abstract question of individualism as compared with communism is one which has at all times attracted to itself the attention of thoughtful minds; and in recent times it has forced itself in many quarters, quite into the domain of practical politics. Whatever attractions, however, this question may possess, it is quite beyond the province of this paper. I shall simply confine my attention to-night to the question as to whether that interference of the community with the liberties and responsibilities of the individual which public medicine has brought into existence within the last quarter of a century has really been doing good.

Let me in the first place draw your attention to the mortality curves which I had the honour of showing to the Society about two years ago. The first shows the mortality for phthisis per 10,000 of population between the years 1860 and 1905, in Aberdeen, Dundee, and Edinburgh. Here there have been no legal enactments at work, only progressive efforts to improve the general conditions of life. The results of these efforts are, as shown by the curves, as satisfactory as they are distinct.

The next set are those of typhoid fever for the same time and in the same towns, and they have on them marks showing the year when legislative enactments stepped in, compelling notification and inducing, more or less thoroughly, hospital isolation. Does the curve indicate

that these enactments have had the effect which was to be expected of them? We must admit, I think, that it certainly does not. As regards measles there is no doubt that the curves show that since notification and strict hospital isolation came in, the mortality has all over increased rather than diminished. As regards diphtheria, the curves show in all the towns improvement, but it will, I think, be admitted that since the period of strict isolation the improvement is not one specially creditable to isolation. At any rate, what improvement there is is not more marked than that shown by phthisis without any isolation at all, or than that shown by typhoid fever before the strict modern isolation stepped in. As regards scarlet fever, the curve shows, since isolation, a distinct improvement. When I read my paper to this Society two years ago, I was under the impression that this was an example of *propter hoc*, and that notification and isolation should get all the credit. I am sorry to say that I now believe it is rather a case of *post hoc*.

In the journal *Public Health* for April 1901, Dr Killick Millard published an article on "The Influence of Hospital Isolation in Scarlet Fever." In this article he compares the incidence and mortality of scarlet fever in certain English towns in which strict isolation of this disease has been practised, with its incidence and mortality in towns in which either its isolation has been less strict or in which no hospital isolation has existed at all. He has shown that all over the country, and for the 30 years, 1870 to 1900, the mortality from scarlet fever has been greatly and progressively diminishing, but that this diminution has been as marked in those towns which had no isolation hospital as in those which had isolation treatment for 70, 80, or 90 per cent of the cases. Dr Millard's conclusions are —

1. That the hospital isolation of scarlet fever in towns appears to have failed to materially reduce the prevalence of the disease.

2. That the hospital isolation of scarlet fever appears to have failed to materially reduce the fatality of the disease.

3. That the fatality of the disease has fallen most in those large towns which have not practised hospital isolation.

In order to discover if any information could be got from examination of the mortality rates in those same towns from other infectious diseases, I obtained from Dr Crawford Dunlop a table showing the mortality in them from scarlet fever, and also from diphtheria, measles, fevers, and whooping-cough, during the 30 years, 1874 to 1903 (see Table I.). Further, seeing that at the present

time all the large English towns, except West Hartlepool, have either got isolation hospitals, or have made arrangements for hospital isolation of their infectious cases, I append Table II., which shows the death-rate per 100,000 from those same diseases in the Yorkshire towns, including of course West Hartlepool, and during the years 1897 to 1906. Whilst a glance at those tables illustrates the very great need for caution in handling such statistics, it must yet, I think, be admitted that the results show that isolation hospitals have not had the effect on the death-rate which the public had the right to expect.

At this moment I shall do nothing more than simply mention the fact that what may be called natural isolation is hardly a condition to be wished for by the community. We all know that the relatively harmless measles or scarlatina of our town districts can become virulent and fatal when given root in outlying regions or countries which have not had these diseases among them for generations. Every town doctor who sees much of the exanthemata recognises that the severest cases are usually those occurring in the country bred who have just come into town. In this connection, I have endeavoured to find out if scarlatina could be shown to run a mild or severe course according to whether the patient had or had not suffered previously from any of the other infectious diseases. I collected about 1000 cases of scarlet fever in patients over the age of 6 years, and classifying them into (1) Mild and moderate, (2) Severe and fatal, I arranged them under the following heads :—

(*a*) Those which had had previously no infectious disease at all.

(*b*) Those which had had previously one, generally measles.

(*c*) Those which had had previously two, generally measles and chickenpox or whooping-cough.

(*d*) Those which had previously had three, generally measles, chickenpox, and whooping-cough.

(*e*) Those which previously had had four, generally those same diseases with diphtheria or mumps added.

The results I have illustrated by curves.

In endeavouring to interpret these curves one has to remember the apparent contradictoriness of everything in nature. For example, we know that as regards nutrition, excess and abstinence are alike causes of disease—as regards reproduction, sterility and over fertility are alike marks of weakness. In curves like these, therefore, we might expect that the severe and fatal cases would tend to show in numbers at the extremes. At the commencement,

TABLE I.—*Death-rate per* 100,000 *from Infectious Diseases in Selected English Towns.*

		SCARLET FEVER			DIPHTHERIA			MEASLES			"FEVERS"			WHOOPING COUGH		
		1874-83	1884-93	1894-03	1874-83	1884-93	1894-03	1874-83	1884-93	1894-03	1874-83	1884-93	1894-03	1874-83	1884-93	1894-03
Early Hospital Isolation	Huddersfield	17	24	17	5	12	14	68	59	27	21	13	13	58	41	32
	Nottingham	24	17	16	5	10	11	46	48	38	41	31	28	40	52	34
	Bradford	13	31	18	8	6	13	44	50	33	36	18	17	55	46	30
	Derby	48	14	14	3	11	9	31	41	33	30	24	19	36	44	28
Late Hospital Isolation	Oldham	24	33	25	11	17	20	48	83	64	41	18	13	64	58	45
	Halifax	22	18	13	6	12	13	19	47	33	67	19	17	19	29	21
	Bolton	13	30	20	7	19	13	80	83	52	29	26	28	51	65	40
	Birkenhead	23	30	19	3	11	27	40	54	45	44	31	28	68	46	45
	Blackburn	46	54	21	1	7	28	87	95	74	69	32	24	42	54	34
No Hospital Isolation	Preston	34	41	18	9	17	17	66	87	69	61	43	29	56	64	44
	Swansea	—	66	12	—	6	4	—	61	43	—	34	14	—	52	43

NOTE.—As notification and hospital isolation were instituted for the most part in the 'eighties, any effect of these on the mortality of each disease should be shown by the comparison of the figures of the second and, still more by the third columns with those of the first.

TABLE II.—*Death-rate per* 100,000 *from Infectious Diseases in Yorkshire Towns, including West Hartlepool.*

	Scarlet Fever.	Diphtheria.	Measles.	"Fevers."	Whooping-Cough.
Rochdale . . .	12	16	38	9	25
Burnley . . .	30	30	67	20	40
Blackburn . . .	28	30	57	20	30
Preston . . .	19	19	86	28	42
Barrow-in-Furness .	12	15	51	18	32
Huddersfield . .	13	12	36	14	26
Halifax . . .	16	16	36	14	18
Bradford . . .	17	21	32	17	24
Leeds	16	33	47	16	35
Sheffield . . .	22	43	52	23	37
Rotherham . . .	22	16	57	20	38
York	11	8	29	21	25
Hull	11	23	44	18	35
Middlesborough . .	17	26	48	26	38
Stockton . . .	13	24	45	17	34
West Hartlepool . .	9	23	28	11	38

because there it would be denoted that the disease organism had got into what was practically a virgin soil—at the end, because it would be denoted that the organism had got into a soil which had feeble powers of resistance to all organisms. On the other hand, one might expect to find that the mild and moderate cases of scarlet fever would show themselves most about the middle of the curve, where there was some inherited resistance power to organisms, and where this had been strengthened or put into good fighting condition by previous struggles with one or two other organisms. Except, however, that the curves show that most of the severe and fatal cases have had one infective disease previously, and that most of the mild and moderate cases have been in individuals who have had two, I do not think that much importance can be attached to it. I have to admit that in making up these curves, the number of severe and fatal cases of scarlet was proportionately so small that this alone may be a source of error.

But now in connection with the hospital isolation of infectious diseases there is another point to which I wish to refer. This is what is known as "hospitalism," and it signifies the risk to the individual from residence in a fever hospital ward. A great many statistics have been collected showing that the mortality from scarlet fever in isolation hospitals is greater than in home-treated cases. One must not, however, attach too much importance to these, as in tabulating statistics of this kind there are many sources of error, an important one being that hospital cases are almost certainly, from heredity and surroundings, more severe cases than home-treated ones. But in connection with this, I wish to put before you that in looking over my own statistics, I find that out of 930 cases of scarlet fever over 6 years of age, 13 were fatal, and of this 13, 3 were cases in which the patient had caught the infection in the wards. Again, that out of 9 cases of infection of scarlet fever occurring in the wards, 3 were mild, 1 was moderate, 1 was severe, and 3 were fatal.

This shows that patients suffering from other diseases run extra risk if exposed to scarlet fever infection. I cannot but think further that for the occurrence of relapses, sequelæ, and complications of scarlet fever, this so-called hospitalism may to some extent be blamed. It seems to me that there is one thing which we should never forget in dealing with infectious diseases. This is that adults and children living at home may be living in what are called insanitary conditions, and among all sorts and conditions of organisms, and yet be safer there than in a hospital. This is simply because, living at home,

they are living among organisms to which they have become adapted, and are therefore in a way immune. Put them, however, into a fever hospital, where they meet with all sorts of organisms and blends of organisms to which they are not accustomed, and they suffer accordingly.

This brings me to the last point in my paper, and that is that I hold most emphatically that we doctors have for years taken up an unscientific and decidedly unphilosophical view of organisms in general. We have regarded them, and I say this with all reverence, as having been specially created, immutable, and unchangeable by Providence, to be a source of trouble and death to man. Nothing is further from the truth. A disease organism is simply a normal organism out of its place or soil, and largely so because the nutritive conditions of its soil or place have been defective. We may rest assured that what is true of the pneumococcus in pneumonia or the bacillus coli in peritonitis, is in a way true of many other organisms. In this way, then, personal infection, although real and important enough, has had given to it a prominence far above its deserts. I am very pleased to see that to this subject attention is being directed more and more. Thus, in the discussion on Dr Killick Millard's paper, previously referred to, Dr Boobbyer said: "Until we know more than we do at present of the life history of the streptococcus scarlatinæ, or whatever the particulate cause of the disease may be, it were perhaps idle to dicuss the possible influence of insanitary and other conditions and surroundings in its propagation. I have myself been witness to the coincidence of dry seasons as noted by Gresswell and Longstaff, and also the disturbance of impure soil on a large scale with outbreaks of unusual magnitude. But whether such agencies are or are not operative in fostering the spread of the disease, the impression grows upon one more and more with the growth of one's experience, that scarlet fever is capable of spreading by other channels than that of personal infection. Still, impressions with more or less disjointed facts to justify them are not of much scientific value. I shall content myself therefore with saying in conclusion that until we know definitely whether the essential micro-organism of scarlet fever is obligate or facultative (either as saprophite or parasite), we cannot satisfactorily decide whether in this disease we have to deal with a simple matter of personal infection, or with a much larger matter in which the phenomena known collectively as scarlet fever are simply an incident."

M'Vail, in his book on the "Prevention of Infectious Diseases,"

after discussing outbreaks of diphtheria as the result of milk from diseased cows, points out the probability of the origin of diphtheria from dirt and manure heaps and from drains.

It is refreshing in those days to hear any one, especially a medical officer of health, admit that epidemics may break out and spread independently of "contacts" and "carriers." And to those who, like myself, have seen smallpox as well as scarlet fever, measles, diphtheria, and typhoid all treated at home, with no harm to any one and relatively little fuss to all concerned, it requires some self-control to leave the infection and isolation bogeys alone. I shall, however, do so, as my object is simply to show that the strict isolation measures of the past 15 or 20 years stand condemned on their own showing, and that distinct modifications are now required. It is hardly necessary for me to add that there is no one who has a higher opinion of the value of public health work and isolation hospitals than I have—the work for the benefit of the community which can be done by them is enormous, and will continue to be so for years. But the functions which in many cases they have been performing is not what is in their own interests, in the interests of the community, or in the interests of the profession. Every thoughtful and experienced doctor will, I think, acknowledge that any disease, if allowed as it were to run riot, tends to become infectious. Some of us, for example, have seen pneumonia run riot in a locality, and become to all appearances infectious. Again, in Russia, in times of privation and famine, when scurvy runs riot, it is, I understand, regarded there as an infectious disease, and justly enough so. Yet, ordinarily, in this country, pneumonia and scurvy can be with absolute safety treated at home, or in a general ward, and the part which infection plays in their onset may, as far as the community is concerned, be neglected. What I would suggest, therefore, as regards the interference of the community with diseases is that the general practitioner, and especially the general practitioner of some years' standing, should now make himself heard. He has hitherto modestly held himself aloof, and I hold too modestly has stood by whilst legislative efforts, of which he could not approve, were being carried forward. The general practitioner is the man who really knows disease, and I believe that if in former times the community had turned to him more frequently for advice it would have been all to the community's advantage.

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